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The R. H. Fetherston Memorial Lecture.[1]

THE FETHERSTONS AND THEIR COLLEAGUES: EARLY HISTORY OF THE ROYAL WOMEN'S HOSPITAL, MELBOURNE.

By COLIN MACDONALD,
Melbourne.

THE names of Fetherston and other Irishmen are important ones in the early history of the Women's Hospital, Melbourne, and give rise to the reflection that Irishmen exerted a really powerful influence on politics and on the professions of law, the church and medicine and on other branches of higher education in Victoria in the last century. The years of rapid development in Victoria were lean years in Ireland; between 1845 and 1865 the population of that country fell from eight to five and a half millions, and the shores of that famished country witnessed one of the greatest exoduses in European history. Thousands of these Irish emigrants came to Victoria; many of them were men of ambition who had been forced out of the overcrowded professions of their homeland. In the first fifty years of responsible government in Victoria there were five Irish-born premiers—Sir John O'Shanassy, Sir Charles Gavan Duffy, Sir Bryan O'Loghlen, William Shiels, and Sir William Irvine—all men of distinction. From 1857 to 1936—a span of seventy-nine years—four Irishmen in turn held the position of Chief Justice of the Supreme Court of Victoria—Stawell, Higginbotham, Madden and Irvine. Add to these Professor Hearn, first Dean of the Faculty of Law in this University of Melbourne, and such distinguished judges as Sir Redmond Barry, Sir Robert Molesworth, Sir Frank Gavan Duffy and Henry Bournes Higgins, and we can sense the magnitude of the Irish contribution to our legal system. Five of the first seven Chancellors of our university were Irishmen—Barry, Stawell, Hearn, Madden and MacFarland.

And Irish physicians and surgeons played a vital role in the medical profession of Victoria in the last century. The list of doctors in Port Phillip in the 1840's contains many Irish names. By the 1860's, Irishmen were prominent in every branch of medicine: Thomas FitzGerald, brilliant in surgery and uncanny in diagnosis; Richard Tracy, gifted in obstetrics, and recognized as leader of his profession; Richard Eades, health officer of Melbourne; Gerald Fetherston, Thomas Black, William McCrea and many others whose names were respected in their generation.

Perhaps there are qualities which tend to make Irishmen good doctors and obstetricians. They do not lack confidence and charm; may we say that "a bit of the blarney"

[1] Delivered at the University of Melbourne on Monday, August 27, 1956.

enhances their bedside manner? It has been written that their aptitude for obstetrics may derive from the domestic squalor of the poorer classes of nineteenth century Ireland; to Irish lads, the sight of the birth of piglets in a corner of a smoky cottage was not unusual. But these conjectures aside, the Irish doctors who came to Victoria in the stream of migration in the 1850's and 1860's seem to have had one very substantial asset—sound training in the medical schools and hospitals of the Dublin of that time. And this is not surprising, because the early and mid-nineteenth century period may well be described as the golden age of Irish medicine, led as it was by such great doctors and teachers as Graves, Corrigan, Stokes and Adams. And European authorities considered that at this time the Dublin School of Midwifery—of which the Rotunda Hospital and its notable masters were the centre—was the best in the British Isles.

One of these Irish medical émigrés to Australia was Fetherston, senior, Gerald Henry Fetherston, the father of Richard Herbert Fetherston, in whose honour this memorial lecture is named.

Gerald Fetherston was born in Roscommon, one of the counties of Ireland's western province of Connaught. As a youth, he acted as assistant to his father, a doctor, to whom he was apprenticed. He studied at Mercer's Hospital and Ledwich Medical School, Dublin, supporting himself the while by working in chemists' shops.

Before qualifying in medicine, Gerald Fetherston went to sea as an assistant medical officer, sailing with immigrants to America and Australia. In 1856, at the age of twenty-seven years, he obtained his licentiate of the Apothecaries' Hall (Dublin). In the next four years Fetherston made nine voyages to Australia, in those famous ships of the Black Ball Line: they were *James Baines*, *Marco Polo* and *Lightning*. He was in *James Baines* when she sailed from Liverpool to Melbourne in the all-time record of sixty-four days, making the return voyage in sixty-three days, but it was from *Lightning* that he permanently settled in Australia in 1860, after having passed the examination for Licentiate of the Royal Colleges of Edinburgh.

I should say something more about these big clipper ships, for on them Fetherston spent the best part of eight years. These ships belonged to James Baines, who as a young man in the 1850's had already won a reputation among the hard-headed Merseyside shipowners, at the time when his widowed mother was still behind the counter of her small sweets shop in Liverpool. On the discovery of gold in Australia, passenger accommodation to this country, no matter how rough and uncomfortable as long as the ships were fast, was immediately at a premium. Until then the Australian trade had, with few exceptions, employed only second-rate vessels. Shrewdly appraising the prospects of the lucrative passenger trade with the Australian gold ports, Baines realized that special ships were essential—ships which not only were fast, but also had hulls sufficiently spacious to carry a large number of passengers and the supply of water that was always their problem. And so he approached Donald Mackay, the great Scottish-American shipbuilder of Boston, and asked him to design and build a fleet of large clippers. From magnificent New England timber, Mackay built these ships of the Black Ball Line, the first ships to be designed and built by Americans for Englishmen since the War of Independence. Of these, the most famous was *Lightning*, the clipper on which Fetherston finally migrated to Melbourne. For many years *Lightning's* career was a record of fast passages, and her best day's run was no fewer than 436 miles—the greatest in the history of sailing ships. In 1869 she was destroyed by fire while loading wool at the pier at Geelong.

In later years, when Fetherston rode on emergency calls in the scrub beyond Melbourne, the sound of the sea coming in on the night wind may well have brought back these memories; and in his mind's eye he saw again these graceful clippers, their decks noisy with immigrants, their sails snow white and perfectly set, scudding across the green wastes of the Southern Ocean.

Soon after arrival in Melbourne, Gerald Fetherston was appointed Resident Surgeon to the Lying-In Hospital, living there for the next four years; his wife became the matron, and his son Richard Herbert was born within the hospital walls in 1864. In this year Mrs. Fetherston resigned as matron, being followed by her sister, Miss Harvey, who continued in this office for another seventeen years.

In 1865 Fetherston gained the degree of doctor of medicine of the University of Melbourne under a special regulation allowing those possessed of a licentiate of any British Royal College to sit for the examination for the bachelor's or doctor's degree in medicine at a single examination. This special regulation continued in force for only a short time, for it was designed to provide a foundation of "M.B.'s" and "M.D.'s" for the then newly created medical school of this university. On leaving the Women's Hospital, Fetherston bought the practice of Dr. Job Phillips, of Prahran, and was prominent in the professional and public life of that district for the next thirty-six years. The Prahran practice, which extended as far as Oakleigh and Mordialloc, was of course worked with horses, and Fetherston had a large stable of five buggy horses and a saddle hack, at night always kept saddled and rugged for emergency calls.

Gerald Fetherston was first elected to the honorary staff of our hospital in 1869 and continued until 1891, when his son, R. H. Fetherston, was appointed.

Amongst Gerald Fetherston's public and professional offices were the following: Health Officer of the City of Prahran, member of the Council of this university, member of the Medical Board of Victoria, and president of the Medical Society of Victoria. He took an active part in building the original Medical Society Hall in Albert Street, subsequently demolished to make way for the present building there. He had a life-long interest in military affairs, and in 1890 was appointed Principal Medical Officer for Victoria. During the South African War, he was responsible for the medical dispatch of the Victorian contingents, and on their return dealt with the wounded and the sick.

Fetherston, senior, died at his home, "Kia-ora", High Street, Prahran, in 1901, aged seventy-two years. In this house his son, R.H., practised for many years, and one of the latter's partners, Dr. R. N. Wawn—himself on the Women's Hospital staff for twenty-seven years, and happily still in active practice—continues to occupy this two-storey brick house, well known as a doctor's residence for almost ninety years.

So much for the father; what of his only son, R. H. Fetherston?

As I said previously, R. H. Fetherston—known always as "Bertie" Fetherston—was born in our Women's Hospital in 1864, and he was one of the brilliant men who have served the hospital both as "resident" and as "honorary".

After attending Wesley College, he went, as a youth of seventeen years, to study medicine in Dublin. Always before him was the example of his father. They were very dissimilar physically, the father tall and broad, the son slightly built. But their interests were closely akin, both inside and outside the profession.

In October, 1881, he sailed for Britain and began the winter session of the School of the Royal College of Surgeons, Dublin, two years later proceeding to the Medical School of Trinity College. He qualified in medicine with the L.R.C.S.I. in June, 1884, only two years and eight months after starting medicine. He was then twenty years and one month old. During the course, he won special prizes in anatomy, surgery and other subjects, and was appointed a demonstrator in anatomy.

Next year he was at the University of Edinburgh, gaining the degree of bachelor of medicine there; he was able to traverse the whole degree examinations in one year. The young graduate then sought permission to sit for the Irish Surgical Fellowship, but this was refused, as the minimum age required was twenty-six years, and he was not yet twenty-three. The winter of 1886-1887 was spent at hospital work in London, and after being abroad for six years, he returned to Melbourne, soon accepting a

position as resident medical officer at the Women's Hospital.

Three years later, in 1892, Bertie Fetherston was elected to the honorary staff. In 1914 he resigned from the Women's Hospital to take the position of honorary gynæcologist newly created at the Melbourne Hospital; and on his retirement from there, in 1924, he had been an "honorary", at one or other hospital, for more than thirty-two years.

Fetherston was a successful and popular clinical teacher and lecturer at both the Women's Hospital and the Melbourne Hospital, and an examiner for the bachelor's and doctor's degrees in medicine for several years. As medical officer at Wesley College, he pioneered, in Victoria, the regular medical examinations of schoolboys. A highly trusted member for many years of the Council of the Victorian Branch of the British Medical Association, he was President in 1911, and later a Trustee of the Medical Society of Victoria, of which his father had been President forty years before. He took an active part in forming the now very valuable Australasian Medical Publishing Company, Limited, and was an original member, becoming a Director in 1917. He also helped in the formation of the Medical Agency and Medical Insurance Company. Fetherston was a Foundation Fellow of the Royal Australasian College of Surgeons, and a doctor of medicine of the University of Edinburgh.

He was under average height, and invariably wore a "billycock" type of hard felt hat—a cross between a "bowler" and a "topper".

Bertie Fetherston was, again like his father, identified with the municipal life of Prahran for many years, for forty-two of which he was that city's Health Officer.

He also had a term, in the mid-1920's, in the State Legislative Assembly, as Member of the Legislative Assembly for Prahran. But he possessed no adroitness as a politician, though he made many friends amongst the members. This, to him, was the only pleasant part of a parliamentarian's life, which otherwise he considered was little worth the trouble, worry, expense and abuse. Like his father, too, he evinced a lifelong interest in military affairs. He volunteered for the Australian Imperial Force on August 5, 1914, was promoted full colonel in 1915, and early in 1916 was made Director-General of Medical Services with the rank of major-general, after his return to Australia from reorganizing the Australian Army Medical Corps abroad, during which time he was on the Gallipoli Peninsula, with Victor Hurley as his staff officer.

R. H. Fetherston died at his home, "Derwent", Saint Kilda Road, Melbourne, in 1943, in his seventy-ninth year, greatly respected by all who knew him. He had two sons and a daughter. One son—the fourth generation of Fetherston doctors—lives in New South Wales.

My late partner, Sir Stanley Argyle, and R. H. Fetherston were friends—amongst other ties, they had represented adjoining constituencies in the State Legislative Assembly—and it was through Sir Stanley that I came to know "Bertie", whom I regarded as one of the great Australian doctors. To many young men he gave help and encouragement in the speciality, and two of these who rose to particular eminence were Arthur Wilson and Roy Chambers—names of happy memory in this city.

And who were the colleagues at the Lying-In Hospital and the Women's Hospital of these Fetherstons—father and son?

Appointed in the Fifties.

R. T. Tracy	1856-1874
John Maund	1856-1858
W. M. Turnbull	1859-1867

Appointed in the Sixties.

L. J. Martin	1867-1878
G. H. Fetherston	1869-1892

Appointed in the Seventies.

Joseph Black	1874-1876
Thomas Rowan	1876-1895
W. Balls-Headley	1878-1900
S. J. Burke	1879-1892

Appointed in the Eighties.

Felix Meyer	1888-1918
M. U. O'Sullivan	1888-1914
J. W. Dunbar Hooper	1888-1898
G. Rothwell Adam	1888-1918

Appointed in the Nineties.

J. J. Prendergast	1892-1895
R. H. Fetherston	1892-1914
Eugene Anderson	1892-1902
George Horne	1895-1920
E. Alan Mackay	1895-1898
T. Taylor Downie	1896-1910
(Sir) George Cuscaden	1896-1917
F. W. W. Morton	1896-1917
Frank A. Nyulasy	1896-1898
R. H. Morrison	1899-1925
D. McM. Officer	1899-1903

Appointed 1900-1910.

Helen Sexton	1900-1910
H. Cairns Lloyd	1903-1924
A. Norman McArthur	1903-1909
R. Tate Sutherland	1909-1927
J. H. Nattrass	1910-1926
Allen Robertson	1910-1931

Appointed 1910-1914.

C. S. Sutton	1913-1914
Arthur Sherwin	1914-1946
Sydney Allen	1914-1937
N. L. Speirs	1914-1948
B. Milne Sutherland	1914-1946
W. G. Cuscaden	1914-1947

Richard Tracy and John Maund.

When Gerald Fetherston came, in 1861, as resident surgeon to the Women's Hospital, the dominant figure there was a thirty-four year old Irishman, Richard Thomas Tracy, who was born in the old city of Limerick. Tracy's early education had been directed towards the Anglican ministry, but at the age of sixteen years he resolved to enter the medical profession, and his love for it never abated. After he had worked for a year or so as a dresser and wardsman in the County Limerick Infirmary (a beginning he claimed to be of much benefit in his after career), his medical studies proper were begun at Trinity College, Dublin. At this time Ireland was ravaged with typhus and famine, and in three summer vacations Tracy volunteered for work with the Irish Board of Health. He himself contracted typhus, and was lucky to survive.

In 1848 he obtained the Licentiate of the Royal College of Surgeons of Ireland, and a year later took charge of a large city cholera hospital in Glasgow, gained the degree of doctor of medicine of that university, and returned to Ireland to manage a dispensary in King's County. He now wished to marry, and the prospect of building up a successful practice in Canada or Australia attracted him greatly. What was it to be, Canada or Australia? His decision was quickly made one evening in 1851 in his brother's rooms in Lincoln's Inn Fields, London; he spun a coin and Australia won the toss. He hastened to Ireland to marry his cousin, and the couple returned to England, and in May, 1851, set sail for Australia, with him as surgeon on the ship *Ballangeich*.

Not long after he had commenced practice in North Adelaide, the gold fever gripped him, and with James Bonwick, the historian, he prospected unsuccessfully on the Victorian goldfields. But the diggings atmosphere did not suit him and he returned to medicine, first in Bendigo, and soon after in Fitzroy; there his reputation so grew that within ten years his unpretentious house—still standing, and now numbered 165 on the west side of Brunswick Street—was said to be as well known as any public building in Victoria. (Tracy's house in Brunswick Street is now housing two Italian families running a sweets shop and milk bar.)

Tracy had been only a short time in Melbourne before his warm sympathy was excited by the maternity needs of the poorer people. In 1856, in association with his friend and neighbour, Dr. John Maund, he established at 41 Albert Street, East Melbourne, the Lying-In Hospital, whose name was later changed in 1884 to the Women's Hospital. For eighteen years Tracy laboured ceaselessly and unselfishly for this hospital, defending it against all critics. The hos-

pital in turn contributed to Tracy's professional success, enabling him to specialize in diseases of women and children and to develop a skill which became famous throughout Australia.

Tracy had great success as a gynæcological surgeon, carrying out difficult operations which he had never seen performed before. Using detailed advice sent out by Spencer Wells, the distinguished London surgeon, he performed in 1864 the first successful ovariotomy in Victoria. In ten years he operated on 22 such patients, and his death rate of 18% was even lower than that of the eminent Spencer Wells. The Obstetrical Society of London recognized Tracy's work by electing him an Honorary Fellow—a distinction then akin to the highly prized Honorary Fellowship of the Royal College of Surgeons. Tracy was president of the Medical Society of Victoria in 1860, and a few years later was appointed the first lecturer on diseases of women and children in the University of Melbourne. He was also a magistrate, a health officer, a member of the volunteer militia and a trustee of Saint Mark's Church of England, Fitzroy. In all these offices, as in his profession, his singular gifts stand out—his Christian faith, his remarkable self-reliance, his warm-heartedness, his strong will and his loyalty to high ideals. He was everywhere acknowledged to be the leader of the medical profession in Victoria.

In 1873, his health enfeebled by a mysterious ailment, he left for a trip to Britain, and with him went the fervent wish of thousands that his health would be fully restored. But twelve months later he returned to his mansion home in Collins Street East,[1] wasted and in great pain, and there he died in his forty-ninth year, on November 7, 1874.

A post-mortem examination revealed that Tracy had died from an abdominal malignant growth, for a long time unsuspected, and in the interests of medical knowledge the *Australian Medical Journal* honoured Tracy's strange but firm request that a full account of his autopsy should be published. Tracy's obsequies, again at his own express wish, were marked by the utmost simplicity; the hearse was plain, the carriages were few and there were no plumes or trappings as were common at the time. But Melbourne has rarely witnessed such a procession of mourners or such a dense crowd as that which lined the hushed thoroughfares from his Collins Street home to the Melbourne General Cemetery.

Tracy's medical collaborator in the Lying-In Hospital foundation was John Maund, who had died two years before Gerald Fetherston arrived in Australia, but whose name in any mention of the hospital's early history merits the highest recognition. Maund was born in the market town of Bromsgrove, Worcestershire, in 1823. His father, Benjamin Maund, was famous throughout Europe as a botanist and author. John Maund served an apprenticeship to a surgeon in Lancashire. He studied medicine in Glasgow, and in his twenty-first year became an assistant surgeon at Saint Pancras Infirmary in London. After continuing his medical education in London and Paris, he gained the degree of doctor of medicine at the University of Saint Andrews and began practice at Harlow in Essex. As he had never been robust, it was thought that the invigorating climate of southern Australia would be beneficial, and he therefore set sail in 1852, a year after the discovery of gold.

Maund had become increasingly interested in analytical chemistry, and had hoped in Australia to practise as an analytical chemist rather than as a doctor. But medical practice in the new suburbs rapidly growing around Melbourne soon absorbed him, though later he did serve as Victoria's first Government analyst.

A sincere Christian, kind and gentle by nature, Maund was perturbed to see the unhygienic conditions in which the poor gave birth to their children. The idea of establishing a Lying-In Hospital captured his sympathy, and he received an enthusiastic collaborator in Richard Tracy. So on August 19, 1856, were admitted the first patients to the commodious house that they had hired at 41 Albert Street. By the end of the year, the hospital had over 20 in-patients and 100 out-patients, to whom Maund and Tracy gave their services free. The Committee of Management, of which Mrs. Perry, wife of the then Anglican Bishop of Melbourne, was the president, appointed Maund and Tracy medical officers to the hospital for life.

Maund did not live to see the opening of the new hospital on its present site at Carlton in 1858. He died on April 3 of that year at his residence in Latrobe Street East, the description of the illness suggesting typhoid fever. He was only thirty-five years old, and he had been in Australia five short years; but his achievement as founder of the *Australian Medical Journal*, and as one of the founders of the Lying-In Hospital, will preserve his memory. To his contemporaries, however, his professional achievements paled beside his own admirable personal qualities, for it was written at his death that "the Colony of Victoria had never before been called upon to mourn the loss of one who had so much distinguished himself by his attainments in every branch of Medicine, and who was not more distinguished for his abilities, than for his kind and amiable disposition, which had endeared him to all who knew him".

In the English parish church of Bromsgrove, his native town, a stained glass window honours his memory. His portrait in oils hangs in the Medical Society Hall in Albert Street, East Melbourne—only a short distance from the original site of the now great hospital which he helped to found.

Tracy and Maund were the medical founders of the Women's Hospital, where their exemplary and dedicated lives will not be forgotten.

The non-medical people who played an equally important part in the hospital's sound inception were led by three persons: Mrs. Fanny Perry, wife of the first Anglican Bishop of Melbourne, the Anglican Dean of Melbourne (Dr. Macartney), and Mr. Richard Grice, a close friend of Tracy's at Saint Mark's Church, Fitzroy. So the Lying-In Hospital in effect was a foundation of the Church of England.

Felix Meyer.

The next of the Fetherstons' colleagues is Felix Meyer, university lecturer in obstetrics and gynæcology from 1914 to 1918, one of the most scholarly men in the history of the Australian medical profession, and possessed of an unexcelled elegance in both the spoken and the written word.

Meyer and R. H. Fetherston—a few years his junior—were close colleagues for over sixty years, their association commencing when they were boys at Wesley College. Meyer was a man with many interests outside and inside the profession, an excellent conversationalist and public speaker, with a beautifully modulated voice, a first-class scholar and exponent of the classics, a profound music lover, an historian and a prolific writer, for both professional and lay journals.

He was the sole resident medical officer at the Women's Hospital for four years (1881 to 1885), and was elected to the honorary medical staff in 1888, retiring thirty years later. He wrote widely on obstetrical and gynæcological subjects, and made the teaching of students an inseparable part of his work. He was president of the Victorian Branch of the British Medical Association in 1894, and twice represented Victoria at overseas medical congresses.

Like R. H. Fetherston, Meyer was greatly devoted to Wesley College, of which he was *dux* in 1875. He was in very good scholastic company there, for his class-fellows included George Syme, first president of the Royal Australasian College of Surgeons, and Samuel Alexander, later the greatest British philosopher of his period, and one of the only two Australians to have been awarded the coveted Order of Merit. To indicate Meyer's brilliance, at the matriculation examination of 1875 he won the exhibition in classics and the exhibition in English, French, history and geography, and was *proxime accessit* in mathematics. He was the author of a history of Wesley College and also of a life of L. A. Adamson, Wesley College's great head-

[1] Tracy's house—at that time described as 190 Collins Street East—stood on the south side of the eastern end of Collins Street, where the Masonic Hall now stands.

master. Meyer was rarely absent from cricket or football matches when Wesley College was playing, and I first saw him as a keen watcher on the river bank when the school crews were training for the head of the river regatta.

From his days as a resident medical officer at the Women's Hospital, when he kept the betting books for two of his honoraries—I hasten to add that such a procedure, of course, would be unheard of today—he was a regular attendant at Flemington and Caulfield; and at the Melbourne Cricket Club, on days of important cricket matches, I would invariably see him in the back row of the members' pavilion. Yet, as indicating the breadth of his humanity and friendship, T. G. Tucker, world-famous professor of classical philology in the University of Melbourne, regarded Felix Meyer as his greatest friend, and as one of the best of classical and literary critics in the country.

Felix Meyer, a man full of kindness and understanding, died in 1937 in his seventy-ninth year, leaving no children. He was married to the second daughter of Professor Nanson, who held the chair of mathematics in the University of Melbourne for the record term of forty-seven years.

> One afternoon I was walking down Collins Street East with Felix Meyer, when the Victorian Scottish Regiment was marching from Parliament House. "You know, Macdonald", he said, "the Scots are proud of the Gordon Highlanders. But some, like myself, are equaly proud of our connection with the Jordan Highlanders."

M. U. O'Sullivan.

An outstanding name in the history of the Women's Hospital is that of Michael Ulick O'Sullivan, a native of County Kerry, who came to Victoria in 1881, three years after qualifying with the licentiate of the Royal College of Surgeons of Edinburgh. He first practised at Numurkah and then at South Melbourne, but in 1885 moved to Collins Street, when in a few years he had raised his reputation from that of a general practitioner to a height in gynæcology that had probably not been reached before in Australia.

I am told that M. U. O'Sullivan was a deft surgeon, with a finish and neatness about his operations that belonged to a master of technique. His eminence is the more notable because in effect he learned surgery unaided. He was the first surgeon at the Women's Hospital to change into a white suit when operating, and the first to wear rubber gloves. He was elected to the honorary medical staff in 1888, four years before R. H. Fetherston, and at the same time as Felix Meyer and Dunbar Hooper; he retired, because of ill-health, in 1914.

During a visit to Ireland in 1905, he was elected Fellow of the Royal College of Surgeons of Ireland, and while he was in Italy the Pope created him a Knight Commander of Saint Gregory, for O'Sullivan was a staunch supporter of his church. He had a beautiful mansion home named "Eildon" on the south side of Grey Street, Saint Kilda, a little above the Saint Kilda junction; of this home he was very proud, and everything was scrupulously maintained and in perfect order. M. U. O'Sullivan married a daughter of Richard Feehan, who owned the Moonee Valley racecourse; the late R. F. O'Sullivan, at one time senior gynæcologist at Saint Vincent's Hospital, was one of his three sons, another of whom is Dr. Brendan O'Sullivan, still in active practice in Victoria Parade, East Melbourne.

J. W. Dunbar Hooper.

That the appointment of a professor of obstetrics in the University of Melbourne preceded those of medicine and of surgery by almost thirty years was due, in large measure, to J. W. Dunbar Hooper, who, though he had left the Women's Hospital staff in 1898, was, in the early twenties of this century, still deeply interested in maternal welfare. In 1924 he was appointed to a Committee of the Council of the University of Melbourne to advise on the best method of implementing the wishes of the Edward Wilson Trust when that Trust gave £10,000 for the purpose of endowing research in obstetrics. Hooper strongly supported the appointment of Robert Marshall Allan, of Brisbane, as the first director of this obstetrical research. Because of Marshall Allan's successful work, the university became convinced of the necessity of appointing a full-time professor, and this was done in 1929; for a period, half of the salary was paid by the Wilson Trust and half by the university. This professorial appointment was a source of the greatest satisfaction to Dunbar Hooper, for it was his enthusiasm and work which were largely responsible.

Hooper was born in India, the eldest son of Sir William Roe Hooper, one-time surgeon-general to the Indian Medical Service. Educated at Epsom, the public school in Surrey, he qualified in medicine at Edinburgh. He arrived in Australia in 1886 and became resident surgeon at the Women's Hospital, where, as a bachelor of amiable personality and a great diplomat, he is said to have quickly captured the entire ladies' committee. At any rate, only two years later he was appointed to the honorary medical staff, at the same time as Felix Meyer and M. U. O'Sullivan. He resigned, for health reasons, after ten years' service.

The Victorian medical profession owes much to Dunbar Hooper. This was especially so when he was secretary of our Branch of the British Medical Association in the four years 1917 to 1920, a period of anxiety and contention, chiefly because of a serious dispute with the lodges and friendly societies. He was president of the Branch in 1924.

Hooper was a kindly, considerate doctor of the "family" type, and it probably was as much for these qualities as for professional skill that he became well known and liked. He had many interests, all of them directly connected with medicine. Noteworthy was his enthusiastic advocacy of the Australian Aerial Medical Services (the "flying-doctor" service), with which, incidentally, the present chairman of our honorary medical staff, Dr. George Simpson, has had a long and honourable association. Hooper was an original member of the Aerial Medical Services Advisory Committee of the Australian Inland Mission, formed in 1927, the leading spirit of which was the Reverend John Flynn. Hooper was largely responsible for convincing the Branch of the British Medical Association of the value of the flying doctor proposals, and for seeing that the organization set up was soundly founded. Hooper, too, was one of a band of enthusiasts who launched the Melbourne Medical Post-Graduate Committee in 1920; it was at first designed, through intensive and practical courses of instruction, to bring those medical men who had served in the 1914-1918 war abreast of up-to-date developments.

It was while Hooper was medical adviser to the staff at Government House, Melbourne, that he met his future wife, a Swiss lady who had come with Sir Henry and Lady Loch as governess to their daughters. The social columns of the day report that the wedding reception in 1888 was held at Government House, and that Sir Henry Loch gave the bride away at Christ Church, South Yarra; amongst the distinguished guests was none other than the Grand Duke Alexander of Russia—of a species now extinct.

But it must not be thought that upper-crust social activities played any part of importance in Hooper's long life, for he was wrapped up in the profession of medicine. In 1924 he was president of the Victorian Branch of the British Medical Association, and in 1927 president of his speciality at the Australasian Medical Congress in Dunedin. He was physician to the Presbyterian Ladies' College for twenty-five years, and medical adviser to the English, Scottish and Australian Bank and to the Bank of Australasia. He died in 1934, aged seventy-four years.

I knew Dunbar Hooper well; he was a loyal, upright and unselfish man, who for all his forty-nine years in Australia was proud to call himself at heart an Englishman.

Walter Balls-Headley.

Walter Balls-Headley, a tall, distinguished-looking, courteous and scholarly Englishman, played an important part in the Women's Hospital for the last twenty-two years of the past century. He possessed the distinction of being the only member of the Women's Hospital staff to have been elected a Fellow of the Royal College of Physicians (London). Born Walter Balls, he entered Peterhouse,

University of Cambridge, in 1858. Before coming to Australia, Balls held resident appointments at both Saint Bartholomew's Hospital and Great Ormond Street Hospital. In 1878 he became a colleague of Gerald Fetherston on joining the Women's Hospital staff. Subsequently he assumed the additional name of Headley, and as Balls-Headley he is remembered.

During the nineties, Balls-Headley was one of the leading gynæcologists in Melbourne, and occupied an important position in Australian medicine generally. He was university lecturer in diseases of women for a decade, and was the author of a well-received text-book on gynæcology. He had the reputation of being a very neat operator, and it was he who performed the first Cæsarean section in Victoria at the Women's Hospital; the operation (on an unmarried woman) was a great success, and the baby was baptised at the hospital, receiving the names of Balls Headley, after approval by the ladies' committee.

After practising for some years in Tavistock Square, London, Balls-Headley retired in 1908, and finally settled in a remote hamlet in the wilds of British Columbia, where he died in 1918, aged seventy-six years. A long-continued search by me to trace any relatives in England or Canada of this distinguished Cambridge man, who brought considerable lustre to our hospital for over twenty years, has proved fruitless.

C. H. Mollison.

Crawford Henry Mollison was appointed in 1892 as pathologist to the Women's Hospital, and continued in that position for no fewer than fifty-five years. This lovable man, until the last of almost cherubic countenance, with rosy cheeks, pleasant smile and soft voice, was held in the highest regard by all who knew and worked with him over two generations. Mollison's name is deeply etched into the history of the Women's Hospital, where he worked so faithfully for so long.

Of necessity a public figure, as Government pathologist and coroner's surgeon, he never sought the limelight. Quietness and simplicity were his dominant personal qualities, and his contribution to the ends of justice and the welfare of his fellows was unique. He was married three times. His second wife was the daughter of T. A. Browne (Rolf Boldrewood), who wrote "Robbery Under Arms", a piece of effective melodrama constructed around the many stories of Australian bushrangers. Mollison died in 1949, in his eighty-sixth year.

Rothwell Adam.

Following Balls-Headley as university lecturer in obstetrics and gynæcology was George Rothwell Adam, who held this important academic post for fourteen years, and whom many contemporaries regarded as the soundest man of this period in the speciality.

Adam was born at Leeds, Yorkshire, the son of the Reverend George Adam, and he came to Australia as a child. He started medicine later in life than most, for after leaving Scotch College he was on the land for several years before going to study medicine at Edinburgh. His first resident appointment was under Sir Halliday Croom at the Edinburgh Maternity Hospital. He returned to Australia, and appointment to the Women's Hospital followed ten years later. His influence at the hospital was profound, and during the twenty-five years until his retirement in 1913, he was responsible for much of its progress. He was held in high respect and affection by patients, students and colleagues, and his lectures have been described as patterns of excellence—logical, well-elaborated and admirably delivered.

The manner in which Rothwell Adam applied the principles of correct ethical behaviour, in both his professional and his private life, had a considerable influence on all—undergraduates and graduates alike—with whom he came in contact. Adam, who died in 1925, was a notable instance of a man who, having embarked on an occupation which proved uncongenial, had the energy and determination to follow out a course in a totally different sphere, eventually to win a well-earned position of eminence.

Dates of Foundation of Melbourne Hospitals.

The years in which Melbourne hospitals were founded are as follows:

Hospital	Date of Foundation
Royal Melbourne	1848
Royal Women's	1856
Eye and Ear	1866
Alfred	1869
Royal Children's	1870
Prince Henry	1876
Austin	1882
Saint Vincent's	1893
Queen Victoria	1896

Dates of Establishment of Clinical Schools.

The Melbourne clinical schools were founded in the following years:

Hospital	Year
Royal Melbourne	1875
Alfred	1900
Women's	1900
Children's	1933
Saint Vincent's	1910
Prince Henry's	1953
Fairfield	1954

Sir George Cuscaden.

The Fetherstons, father and son, served the Women's Hospital for forty-nine years, but this long and noteworthy father-son association with the hospital was exceeded by Sir George Cuscaden and his son, Dr. William Cuscaden, who between them spanned fifty-one years' continuous service—from 1896 to 1947: in both cases—Fetherstons and Cuscadens—a unique record for one institution.

George Cuscaden was born in Wexford, Ireland, and qualified at Edinburgh in 1880, coming to Australia two years later. He was a man of wide interests, in the forefront of any health movement of importance, an alderman of the City of Melbourne, and Commissioner of the Metropolitan Board of Works. In 1917 he succeeded R. H. Fetherston as Director-General of Medical Services of the Australian Military Forces, and attained the rank of major-general. Over many years he gave worthy service to the people of Melbourne, Victoria and Australia, and well deserved his title of Knight Bachelor, awarded in 1923.

Sir George Cuscaden, aged seventy-five years, died in 1933, and his son, Dr. William Cuscaden, only a few weeks ago; it was the latter who pioneered the modern treatment with radium at our hospital, and he did this with great success.

Reginald Morrison.

Reginald Morrison was chairman of the hospital staff when I came as assistant radiologist to the Women's Hospital in 1925. Educated first at Geelong College, which his father, Dr. George Morrison, had founded in 1861, he studied medicine at Edinburgh, graduating in 1888. His appointment to our honorary medical staff followed in 1899, and in 1920 he succeeded Felix Meyer as university lecturer in obstetrics and gynæcology. Morrison was associated with the formation of the Royal Australasian College of Surgeons, and sat on its Victorian State Council for several years.

He came of a distinguished Scottish-Australian family. His uncle was Dr. Alexander Morrison, principal of the Scotch College, Melbourne, for forty-seven years, and his eldest brother, Dr. George Morrison, was the famous "Chinese" Morrison, adviser to the Chinese Government and an outstanding authority on that country. Another brother was Norman Morrison, who succeeded his father as principal of Geelong College, and one of his sisters was the gracious wife of Mr. Justice Henry Bournes Higgins.

As a young man "Reggie" Morrison was a first-class athlete, excelling at cricket, football and swimming and as a middle-distance runner. He played senior football for Geelong for two years while at school, and when in Edinburgh represented Scotland in international Rugby during three seasons.

Morrison was a tall, good-looking, well-proportioned man of erect carriage, and his photograph in our hospital "Book

of Remembrance" depicts him wearing an emblem of gentility of fifty years ago—the frock coat, which is defined as "a body-coat for men, usually double breasted, having skirts reaching to the knees". Frock coats are not seen nowadays; and yet there must still be hundreds, scarred perhaps by silverfish, secreted in the Olympian fastnesses of Toorak.

"Reggie" Morrison held firmly to high ideals for the profession of medicine. On one occasion I well remember him eloquently propounding that section of the Hippocratic oath which reads: "To hold my teacher in the art equal to my own parents; to make him partner in my livelihood; when he is in need of money to share it with him; to consider his family as my own brothers, and to teach them this art, if they want to learn it, without fee or indenture." He believed the heritage of our profession would be greatly weakened if the father-son or grandfather-father-son tradition in medicine was not maintained and encouraged. "Reggie" was recognized as one of the best bridge players in Melbourne, and it was unwise, when partnered with him, to lead from "King and another". He could, when the occasion demanded, exhibit a severe exterior, and his tongue cut like a lash; but underneath lay a very kindly heart. He died in 1941, aged seventy-six years.

Helen Sexton.

Helen Sexton was the first woman appointed to the honorary medical staff at the Women's Hospital. On matriculating from Carlton Ladies' College (long since defunct), Helen was anxious to become a doctor; but her mother and the university opposing any idea of a medical training, she commenced, in the early eighties, an arts course. After her mother's death, Helen met Miss Lilian Alexander, also anxious to study medicine. The two young women decided to approach the University Council, but first looked around for reinforcements. To this end, early in 1887, they put a notice in the daily Press asking any young ladies desirous of studying medicine to get in touch with them. Six responded, and a good deal of publicity was given to their cause. Helen Sexton and Lilian Alexander interviewed each member of the University Council personally, so that when Dr. Alexander Morrison, of Scotch College, moved "that the Council approve of the admission of ladies to the degrees in medicine", the motion was easily carried. The following year, seven women, including Helen Sexton, commenced the medical course, and in her own words "they attended all lectures at the University and hospital work with the men students, the only difference being that they dissected in a small room apart from the men".

Helen Sexton graduated bachelor of medicine in 1892, at the age of thirty-one years, her entrance to the profession being preceded by that of Clara Stone and Margaret Whyte. It was Margaret Whyte who came to the Women's Hospital in 1892 as the first woman resident medical officer, and Amy Castilla followed.

In 1896, with five other medical women under the leadership of Clara Stone, Helen Sexton helped to found the Queen Victoria Hospital, and in 1899 she was appointed to the Women's Hospital as an honorary gynæcological surgeon. She quickly developed a large practice. I have been told that she had a kindly, though somewhat brusque, manner and a broad sense of humour. In 1910 she resigned from the Women's Hospital staff, and two years later went to live in Europe.

Soon after the outbreak of the first World War, Helen Sexton, though this time aged over fifty years, took a small field hospital to France, the gift of herself and other women doctors. She served in the French Army with the rank of *majeur*. In 1917 she came back to Australia, returning to England in 1919. She then finally retired from all practice and afterwards spent several years in Florence, Italy. She was a keen art connoisseur.

During the last decade of her life, she was almost totally helpless with arthritis; but it has been written that "her splendid smile still shone, and the few words she could speak, were always concerned with somebody else's well-being". Helen Sexton died in London in 1950 in her ninetieth year, forty years after her resignation from the Women's Hospital because of an allegedly "bad heart".

Robert Marshall Allan.

R. H. Fetherston had left the Women's Hospital fifteen years before Marshall Allan joined the honorary medical staff on his appointment as the first professor of obstetrics. But the two men were colleagues through sitting together for twelve years on the Council of the Victorian Branch of the British Medical Association. Fetherston was president in 1911, and later became a trustee of the Medical Society of Victoria, thus remaining *ex officio* on the Branch Council. Marshall Allan, on coming to Melbourne from Brisbane, was soon elected to the Victorian Branch Council, and became honorary secretary and later president. Fetherston and Allan were drawn to one another by similar interests, and became close friends.

Professor Allan was born of Scottish parents in Brisbane, and received his early education at the Scots College, Sydney, later proceeding to Edinburgh, where he graduated in 1910. A keen interest in obstetrics and gynæcology was already aroused, and he went to Dublin, to the Rotunda Hospital, where Fetherston had worked twenty-five years before. Awarded the McCosh travelling bursary of the University of Edinburgh, Allan spent a year on the Continent at Paris, Berlin, Munich and Vienna before returning to the Rotunda Hospital to become assistant master under the eminent Henry Jellett. He held this position for three years until the outbreak of the first World War, and for five years Allan was on active service with the Royal Army Medical Corps. He served in the successful campaign led by General Maude for the relief of Kut-el Amara on the Tigris, and was awarded the Military Cross. On returning to Australia, he practised for some years in Brisbane, and in 1925 was appointed Director of Obstetrical Research in Victoria, four years later being appointed professor.

Allan was a handsome man, of well-proportioned build, and with twinkling eyes that attracted attention in any company. He dearly loved the people of the United Kingdom and their institutions, and was never happier than in cementing professional and personal ties with the homeland and New Zealand. Thus the furtherance in Australia of the Royal College of Obstetricians and Gynæcologists was dear to his heart, and its firm establishment here afforded him great satisfaction. He held strong and unequivocal views on the controversial subjets of politics, religion and international affairs, sometimes most forcibly expressing them in unexpected places; but this seeming rigidity of opinion was really only a very translucent façade for his true qualities of sincerity, constancy, helpfulness and kindness, which many a student—graduate as well as undergraduate—had occasion to confirm when examination results were posted. In his desk at the Women's Hospital was found the counsel: "Let me not live after my flame lacks oil to be the snuff of younger spirits."

An omnivorous reader, with a splendid memory, he must surely have been one of the most knowledgeable men of his time in this country, though his wide acquirements were veiled by modesty and often shyness. Pretension and snobbery—intellectual and social—in whatever guise, he was quick to denounce at any time or place. He was steeped in the history and tradition of amateur Rugby football, which he had played well in his younger days. I can well remember how (during the last month of his life), on his being subjected at luncheon to gentle badinage when it was reported that a Rugby player had had an ear bitten off in Brisbane, with what gleeful zest—sick man though he was—he countered by saying that this malfeasance had occurred under the League, and not under his beloved Union code of Rugby rules. Aged sixty years, he died in July, 1946, as he would have wished—at work till the last.

Marshall Allan must be accorded an honoured place in the history of the Women's Hospital.

Lectureships.

Lecturer in Obstetric Medicine and Diseases of Women and Children.

Richard Thomas Tracy	1864
Lawrence Joseph Martin	1875
James Jamieson	1877
Walter Balls-Headley	1889

Lecturer in Obstetrics and Diseases of Women.

George Rothwell Adam	1901

Lecturer in Obstetrics and Gynæcology.

Felix Meyer (Acting)	1906
George Rothwell Adam	1907-1913
Felix Henry Meyer	1914
George Horne	1919

Independent Lecturer in Obstetrics and Gynæcology.

Reginald Herbert Morrison	1920
Arthur Mitchell Wilson	1925

Professor of Obstetrics.

Robert Marshall Allan	1929
William Ivon Hayes (Acting)	1945
James Walter Johnstone (Acting)	1948

Professor of Obstetrics and Gynæcology.

James Walter Johnstone (Acting)	1950
Lance Townsend	1951

Conclusion.

Down the years, a fine band of medical men have served this State: humble bush doctors who operated skilfully, by the light of a hurricane lamp, in the heat of a surgery of corrugated iron, or in the cold of a canvas hospital; surgeons who worked tirelessly in the crowded hospitals of the cities; men like Henry Handel Richardson's "Richard Mahoney" who fought on, though their souls were ever restless in an alien land; doctors who advanced the knowledge of the world in ill-equipped laboratories, when the fates seemed pitted against them; men who worked quietly and selflessly, unnoticed outside their own small realm, saving life and restoring health. There were great men in this band, and their qualities of spirit and mind can still inspire us. In this tradition the names of Gerald Fetherston, his son R. H. Fetherston, and their contemporaries should be remembered, and remembered with honour, particularly by those of us at present enjoying the fruits of their labours.

Within tonight's time limitation, I have been able to say something about only a small percentage of our former medical staff, and this I greatly regret, for some of them, like John Sidney Green and Charles Cunningham, were amongst my oldest friends.

Happily the lives and work of all are now permanently recorded in our hospital "Book of Remembrance" compiled for this centenary.

OBSERVATIONS ON THE VALUE OF THE COMPLEMENT FIXATION TEST IN THE DIAGNOSIS AND MANAGEMENT OF AMŒBIASIS.

By BRUCE HALL AND H. L. CARRUTHERS,
Sydney.

PART I.

(Bruce Hall.)

SINCE the complement fixation test was first introduced by Craig in 1927, there has been considerable controversy as to its value.

For some years past the test has been performed on a large number of patients at the Repatriation General Hospital, Concord. Smaller numbers of patients have been similarly examined by Dr. T. C. Backhouse, at the School of Public Health and Tropical Medicine and more recently at Saint Vincent's Hospital, Sydney.

The antigen used at the Repatriation General Hospital, Concord, and at Saint Vincent's Hospital has been one or other of two products prepared by two American firms. One preparation was used between 1949 and 1952, the other since 1952. Dr. Backhouse has also used antigen which he has prepared himself.

This report concerns a section of the patients examined at the Repatriation General Hospital, Concord. At the moment it is not possible to complete a more comprehensive review of a larger number, which may be published later.

When the work was commenced we tended to concentrate on recently proven cases of the disease, and on such patients as had had a previously proven infection and still exhibited strong clinical evidence of continued activity. Later we included cases never proved by recovery of the organism but providing clinical evidence (usually including sigmoidoscopic evidence), and still later a wider variety of cases. This last inclusion was deliberately made, less to exclude the remote possibility of amœbiasis than to explore possible fallacies in the test, and we included, for example, cases of non-specific ulcerative colitis, carcinoma of the colon, functional gastro-intestinal disorders and so forth. Furthermore, serial observations of the test have been carried out on a number of patients undergoing anti-amœbic treatment.

It should be added that at all times we have been obliged to be careful in the use of antigen, as at times our supply has seemed precarious. Observations in this communication are passed on 206 patients examined by the complement fixation test (*Entamœba histolytica*), divided into the following four groups:

1. Patients with recently proved and active infection ("carriers" excluded)—group referred to elsewhere as "+++".

2. Patients with a previous history of proved infection and still exhibiting clinical evidence of disease (organism not recently recovered)—"++".

3. Patients who had never had a proved infection, but who at some time had been suspected by other observers, or whom we considered reasonably suspect on clinical grounds (commonly having sigmoidoscopic evidence of infection)—"+".

4. Sundry patients only remotely suspect, or suffering other disorders not suspect, and examined in the search for fallacies in the test—"sundry group".

In the first analysis we divided all 206 clinical histories into two groups—(*a*) those in which, at the time of the initial test, it might be anticipated the result would be positive, and (*b*) those in which it was similarly anticipated that the initial test result might be negative. Such anticipations were made on a combined consideration of stool findings, previous history, existing symptomatology and clinical findings (including sigmoidoscopic findings)—that is to say, on a summation of the whole picture at the time of the test. The 206 cases were accordingly grouped as follows, with these results: (i) anticipated positive result, 82 cases; actual positive result, 49 cases; negative result, 33 cases; (ii) anticipated negative result, 124 cases; actual negative result, 105 cases; positive result, 19 cases. In considering the results of this analysis, we paid particular attention to the 19 cases in which positive results were obtained when negative results had been anticipated; we did so because we thought that these results must raise doubts as to whether the anticipated results were wrong because of wrong assessment of the case, or because of non-specificity of the test. Reexamination of these 19 clinical histories revealed the following findings. Nineteen out of 124 patients gave a positive response to the complement fixation test when it had been anticipated that the result would be negative. In five cases the organism was found in the fæces (three patients were asymptomatic "carriers"; in two the symptoms were initially regarded as not due to the *E. histolytica*). Three patients had previously suffered a proved infection. Ten had previously been suspected by other observers of having amœbic disease. One result could not be accounted for at all. That

is to say, out of 124 cases the result was apparently fallacious in one and possibly fallacious in 11. These results at least suggest that the test is reasonably reliable in the matter of specificity.

From the total 206 cases, we selected for closer analysis 103 records which included only (*a*) patients with recently proved and active infection, (*b*) patients with a history of previously proved infection and still exhibiting clinical evidence of the disease, (*c*) patients who had never had a proved infection, but who had at some time been suspected by other observers, or who we considered were suspect on clinical grounds—that is to say, that group initially labelled "+++", "++" or "+". This group of 103 cases was analysed from several different angles, as follows.

The complement fixation test produced positive results in 65 and negative results in 38.

The serological and sigmoidoscopic findings in relation to the stool findings are shown in Table I.

TABLE I.

Serological and Sigmoidoscopic Findings in 103 Cases on the Basis of the Results of Stool Examination.

Parasites in Stools: Present or Absent.	Serological Findings.	Sigmoidoscopic Findings.
Present, 25 cases.	Positive, 17 cases. Negative, 8 cases.	Positive, 20 cases. Negative, 5 cases.
Absent, 78 cases.	Positive, 48 cases. Negative, 30 cases.	Positive, 51 cases. Negative, 21 cases.

The results of the complement fixation test and the sigmoidoscopic findings are shown against the background of the case history in Table II.

TABLE II.

Case History.	Number of Cases.	Serological Findings.	Sigmoidoscopic Findings.
+++	30	Positive .. 18 Negative .. 12	Positive .. 18 Negative .. 12
++	9	Positive .. 5 Negative .. 4	Positive .. 5 Negative .. 4
+	64	Positive .. 42 Negative .. 22	Positive .. 47 Negative .. 17

Although the serological and sigmoidoscopic findings are closely similar numerically, the positive and negative findings did not agree in every case, as will be seen from the following tabulation:

Sigmoidoscopic findings positive and serological findings positive, 41.

Sigmoidoscopic findings positive and serological findings negative, 30.

Sigmoidoscopic findings negative and serological findings positive, 24.

Sigmoidoscopic findings negative and serological findings negative, 8.

In 68 of 103 cases, either the sigmoidoscopic or the serological findings were positive, or both were positive.

Examination of the histories of 73 cases suggested that there was little or no likelihood that the patient had amœbic disease. These were analysed with the following results.

Seventy-three patients, who were not expected to give a positive result, were examined by the complement fixation test. A positive result was obtained in five cases. In one case cysts were found in the stools, but the symptoms were regarded as not of amœbic origin. Three patients had cysts in the stools; they were asymptomatic "carriers". One patient could not be accounted for. In the remaining 68 cases the result of the complement fixation test was negative. The conditions found are set out in Table III.

The following are progressive observations of the results of complement fixation tests on 60 patients undergoing treatment for amœbic disease. They are considered in four groups.

TABLE III.

Condition.	Number of Cases.
Non-specific ulcerative colitis ..	13
Peptic ulcer ..	10
Functional gastro-intestinal disorders of various type ..	28
Crohn's disease	2
Carcinoma of colon	2
Polyposis coli	3
Diverticulosis of colon ..	2
Cirrhosis of liver	3
Hydatid of liver	1
Sundry	4

Group I comprised proved cases of amœbiasis in which trophozoites or cysts had recently been recovered from the stools, and in which there was clinical evidence of active disease. There were 17 cases in this group and the result of the complement fixation test was positive in 14 and negative in three. The courses of treatment given were as follows: one course was given in nine cases, two courses were given in two cases, more than two were given in five cases, and no treatment was given in one case. The results of the complement fixation test before and after treatment were as follows: positive becoming negative, 13 cases; positive remaining positive, one case; negative remaining negative, two cases; negative becoming positive, one case.

Group II comprised cases of previously proven infection with sigmoidoscopic evidence of the active infection, the organism not having been recently recovered. There were 25 cases in this group, and the complement fixation test gave positive results in 21 and negative results in four. The courses of treatment given were as follows: one course was given in 12 cases, two courses were given in seven cases, more than two courses were given in five cases, and no treatment was given in one case. The results of the complement fixation test before and after treatment were as follows: positive becoming negative, 18 cases; positive remaining positive, three cases; negative remaining negative, four cases; negative becoming positive, no case. (Three of 18 patients whose complement fixation test result became negative were examined one year or more later, when they had further symptoms and the complement fixation test result was again positive.)

Group III comprised cases not previously recognized, but diagnosed on the history and clinical findings, including positive sigmoidoscopic findings, the organism not having been recovered from the stools. There were 15 cases in this group, and the complement fixation test gave positive results in 13 and negative results in two. The courses of treatment given were as follows: one course was given in six cases, two courses were given in five cases, and more than two courses were given in four cases. The results of the complement fixation test before and after treatment were as follows: positive becoming negative, 13 cases; positive remaining positive, no case; negative remaining negative, two cases; negative becoming positive, no case. One patient who gave the result "positive becoming negative" was examined one year later; the complement fixation test result was positive, but there were no symptoms or signs.

Group IV comprised incidental cases in which treatment was still continuing. In one case of pyrexia of unknown origin with hepatomegaly, and in a case of unexplained recurrent diarrhœa, the positive response to the complement fixation test became negative during treatment.

In the whole 60 cases of amœbic disease, the overall results of the complement fixation test may be summed up as follows: positive changing to negative, 47; positive remaining positive, four cases; negative remaining nega-

tive, eight cases; negative becoming positive, one case. Three patients examined again one year or more after the completion of treatment again gave a positive response. It was not possible to follow up the whole 60 patients.

PART II.

(H. L. Carruthers.)

There were two questions raised by the results of the serum examinations in this study. The first was concerned with positive serum reactions obtained in cases in which a negative result could reasonably be expected. The second was one raised by obtaining negative responses from serum which could reasonably be expected to give positive results.

In order to assess the first, it was necessary to know the total number of patients whose serum had given a positive result, who were included among the 103 of the chosen series, and who had a normal sigmoid and rectum at each examination. These may be set out as follows:

Negative sigmoidoscopic findings with positive amœbic complement fixation reaction: 24 of the 103.

Proven cases	9
Probable cases	3
Possible cases	12

In 10 of these 12 "possible" cases the stool findings were negative on repeated testing.

It was obvious, therefore, that there were 10 patients with negative sigmoidoscopic findings and negative stool findings and who were in the "possible" category, who had given a positive response to the serum test.

There could be no guarantee that these 10 were giving non-specific results by serum testing, for only the sigmoid and rectum had been visually observed. There may have been lesions elsewhere in the large bowel in these cases, or other evidence of amœbiasis earlier and not easily determined. In the clinical section Dr. Hall has indicated that in only one of 73 remotely possible cases had the complement fixation test produced a positive result, and this is a useful control series.

In the remaining two of the 12 "possible" cases *Entamœba histolytica* was present in the stools; this helps to explain the definition of this category of "possible"—that is, "having a history not inconsistent with amœbic infection, but the disease not established previous to the investigation". Further evidence of the significance of this category is given by the fact that out of the 103 cases selected for analysis by all methods of examination, 64 were listed as "possible". In 42 of these positive serum reactions were obtained, and in 47 positive findings were obtained on sigmoidoscopic examination.

For the various reasons given it would seem a reasonable assumption that the majority of the 10 positive serum findings were actually obtained in cases of amœbiasis, and therefore were specific in nature.

So far as the second question is concerned, at some time early in the first half of the investigation finishing in December, 1954, and quite unknown to me then and since until 1955, Dr. Bruce Hall had some misgivings concerning negative results to the complement fixation test arising from the use of the technique adopted for the examination of each specimen of serum. These negative findings had been obtained in some cases in which, as judged by clinical stool and sigmoidoscopic examinations, there was a good chance that the results would be positive.

There were several possibilities which could have explained this, which are perhaps a little more obvious now than several years ago when this work commenced. The negative findings at any time could have been caused by a failure to produce any antibody whatsoever or only a very small quantity of it. They could have been explained by some qualitative difference in the nature of the serum antibody produced, and would therefore have differed in some way from the antigenic structure of the strains of *E. histolytica* used in Baltimore, United States of America, by the workers of the manufacturing firm Hynson, Westcott and Dunning.

There was a possibility also that the antibody response had failed to coincide entirely with the proven, probable or possible categories selected for examining the findings, so that in those cases in which a smaller amount of antibody was produced there could have been an excess of complementary guinea-pig serum activity in the dose used for each test. This could leave enough remaining over, after the antigen and antibody reaction had completely occurred, to cause lysis of the sensitized sheep's red blood cells. This could have resulted in a "false-negative" report to the physician. There is a disadvantage with a very sensitive testing of sera, in so far as the smaller amount of complement to be fixed may result in non-specific reactions. The many reasons for this will be given later.

Dr. Clive Backhouse compared our first American antigen from Hynson *et alii*, of Baltimore, United States of America, with one prepared by himself and his staff from several strains of *E. histolytica* isolated in culture from ex-service personnel who had been in the Malayan, Polynesian, Melanesian and Australasian regions. These two antigens were tested against sera collected from Dr. Hall's cases and comprising the series of this investigation. The work was carried out independently at the Department of Parasitology, School of Public Health and Tropical Medicine, University of Sydney. Sixty-one sera were examined in all, and the same reaction for both antigens was given by 40 sera only. Some 12 sera reacted with the Australian antigen but not with the American, and nine reacted with the American antigen but not with the Australian.

It appeared likely that Dr. Backhouse had obtained satisfactory serological evidence to explain the anomaly of these early findings; but there could have been other reasons for them, both then and at this time, when more information has been collected, and a second antigen of Ely Lilly and Company (U.S.A.) has been used extensively. The findings pertinent to this difficulty may be stated as follows:

Sigmoidoscopy findings positive: 71 of 103 cases.

Amœbic complement fixation result positive: 41 cases.

Amœbic complement fixation result negative: 30 cases.

Stool findings.

Nine proven cases: one positive, eight negative results.

Three probable cases: No positive results, three negative results.

Eighteen possible cases: Six positive results, 12 negative.

In 12 proven and probable cases, and in approximately two-thirds (12) of the 18 possible cases, a positive result could have been expected; this makes 24 in all, with a maximum of 30.

There did not seem any doubt that a careful examination of all our experimental data was required. It seemed necessary also to restate the categories of the clinical cases. The latter statement has been given first, and this is followed by a description of the general precautions adopted during the investigation. The reasons are also stated for the taking of certain precautions against reactions which could be given by disease conditions other than amœbiasis or amœbic disease, or against those produced by some laboratory mishap or by chemical and physico-chemical happenings.

The Histories and Clinical Findings, and the Categories Adopted for the Analysis of the Results.

In view of the time that might be required for a positive serum reaction, and in accordance also with the clinical severity, a division of the cases was made into two major groups in March, 1949, when this investigation commenced.

The first group consisted of cases in which amœbic disease had already been proved to be present. In some of these active infection was present then (and subsequently), and they were called "proven". A second subgroup was made to include cases in which, although infection had previously been proved to be present, there was no active infection at this time (or subsequently), and therefore these have been called "probable".

The second major group consisted of cases in which up till this time there was only a suggestive history of amœbic disease, but no proof of any sort that it did indeed exist.

Cases of the first group could be expected to present a more reasonable chance of a positive serum finding at this time, although for the "possible" cases the chances might have been improved subsequent to the beginning of the investigation, because of more manifest disease and a better chance of actual proof. The results to be explained will indicate that this probably did happen.

Therefore, for the purpose of description only, for convenience of presentation and as far as practicable with allowance for many variables, the three categories in which the cases have been arranged concern the infective state in the period remotely and immediately before, and at the commencement of, the investigation in March, 1949, and subsequently as far as possible until December, 1954. They have been arranged in three groups, as follows: (i) patients having proven infections before coming to this investigation and still showing symptoms during it ("proven"); (ii) patients having previously proven infections, but with no symptoms or with symptoms not diagnostic of amœbic disease during it ("probable"); (iii) patients having a history not inconsistent with amœbic infection, although the disease was not established previous to the investigation ("possible").

The 73 patients with a condition not likely to be due to amœbic infection, but worth investigating, were regarded as a fourth group and labelled "remotely possible" or "unlikely to have amœbic disease".

In this way an attempt has been made to indicate the type of amœbic disease syndrome or the chances of its being active which were considered during the trial of the amœbic antigens, and whilst positive results were obtained from their use in the complement fixation test, carried out repeatedly on the serum of each patient over the whole time.

The Precautions Observed During the Investigation, and Technical Notes on the Complement Fixation Test.

Many comprehensive precautions were taken during the investigations which it was hoped would be conducive to a more satisfactory idea of the use of the complement fixation test in amœbic disease. They may be set out as follows.

1. During the time of the investigation from March, 1949, until August, 1955, 1115 tests on serum were made and the results reported, without knowledge of the cases considered in the clinical paper. All tests were made with the American antigen of Hynson, Wescott and Dunning at first, and later with that of Ely Lilly and Company.

2. The technique used was in the care of one person during weekly testing for the whole period, and has been kept constant during the examination of an average of 5·36 serum specimens for each case. All specimens of pus or mucus were collected through the sigmoidoscope and examined immediately for trophozoites of *E. histolytica* and for cell exudate or crystals. Stools were examined with never less than six specimens on any one occasion, and during the whole period of inquiry each patient had a large number of tests performed.

3. Apart from the usual routine of examining a specimen of fæces in normal saline, in triple strength iodine and in one in 100 eosin solution, examination has been made after zinc sulphate concentration. Any specimens which appeared to contain cysts of *E. histolytica* or *E. coli*, or any cysts at all, have been reexamined by Dr. Clive Backhouse at the School of Public Health and Tropical Medicine, University of Sydney. He has used iron-hæmatoxylin stain and culture methods if there was any doubt about reaching an opinion. A tap-water suspension of fæces has been used to demonstrate the chromidial bars of *E. histolytica* cysts.

4. A comparison was made by Dr. Backhouse of Hynson, Wescott and Dunning antigen against one prepared by himself from entamœbæ in local cases. It was apparent that the American antigen was of limited reactivity and likely to fail to react with some sera which gave positive results with other methods.

5. A small series of sera were examined in parallel at Saint Vincent's Hospital by the senior technician, Mr. H. Clark, and also at the Repatriation General Hospital, Concord, by us. Both used the same technique developed for this investigation.

Laboratory technical precautions were taken to avoid non-specific serum reactions as far as possible, and to this end there were two major considerations when the complement-fixing test of this investigation was being designed. The first was concerned with avoiding non-specificity arising from the antigen alone or from the serum. The second was concerned with the delicate and colloidal nature of the reagents to be used in the presence of sodium chloride electrolyte, and with the results given. These antigens were experimental and the antibody was an uncertain quality or quantity; we were using the in-vitro activity of complement fixation only, and there was little knowledge of its activity *in vivo* in order to make true interpretations of the results of serum tests.

In the first consideration there was a possibility that the antigen might fix complement without antibody, and to avoid this a suitable dilution with normal saline was used which would give a positive result. It could happen also that the serum might fix complement without antigen, and this could arise from several causes, such as the following: (i) anticomplementary activity of serum despite inactivation by heating; (ii) infection of serum specimens; (iii) unclean glassware or dropped tobacco ash; (iv) hæmolysed serum specimens; (v) the storage in glass tubes of serum separated from the clot; (vi) possibly centrifugation.

One part of fresh guinea-pig serum complement added to 19 parts of normal saline was used throughout, and this approximated three minimal hæmolytic doses in the ordinary way of testing. This dose was used for the first year, and four or five minimal hæmolytic doses were used subsequently. This is reasonably large, and is used in order to guarantee some certainty of a positive result in the Wassermann test. The activity of this complement dilution was measured by the time required to cause 100% lysis of a 3% suspension of sensitized sheep's cells in the absence and presence of amœbic antigen. This usually required four to six minutes. At the final testing of each patient's serum hæmolysis was allowed to proceed at room temperature after all tubes had been placed in the water bath at 37° C. for one to two minutes only. Positive or negative complement fixation responses were determined by observing every tube of each test for thirty minutes and recording the presence or absence of hæmolysis. At the beginning of the investigation serum antibody titrations were made, but later, because of the limited amount of American antigen available, only a fixed dilution of one part of each patient's serum and four parts of normal saline was used for both a control and a testing tube. Weak reactions of inhibition of lysis at this dilution are unreliable and require considerable caution in the interpretation of positivity. A 0·5 millilitre volume of all reagents was used, and a final one of two millilitres in each tube at the last stage of both the titration of complementary activity and of every test made on the serum of patients with known or suspected acebiasis.

The second major consideration concerned the provision of a constant protein concentration for guinea-pig complement and each patient's serum in all tests performed, and it was hoped that this would tend to maintain stability of serum colloids and also that of antigen. To do this it was not possible to titrate a minimal hæmolytic dose of complement on each occasion and so provide a single volume in each test of a fixed number of minimal hæmolytic doses of complement. The titres of complementary activity vary with the collected sera of different guinea-pigs, which produce varying amounts of serum protein on different days of testing. Only "Analar" sodium chloride was used, of minimal impurity, and distilled water was drawn from the same still of tin-lined copper and glass.

To this same end, all specimens were collected by sterile syringes into sterile glassware and refrigerated at 4° C.,

and the serum was allowed to form by natural clot retraction without centrifugation and was removed only on the morning of each day of testing. Inactivation of serum was made for forty-five minutes at 56° C., in a dilution of one in five with normal saline, and the tests were carried out as described. This amount of heat is able to remove complementary activity, to diminish natural hæmolysin for sheep's red cells and to reduce the frequent anti-complementary activity of unmodified and otherwise natural human serum.

Conclusions and Comments.

This test appears to be a reasonably specific one. This finding is in accord with those of a number of other observers.

The test is a useful one from a diagnostic viewpoint, so long as it is used in conjunction with other diagnostic criteria. A positive response to the complement fixation test should not be regarded as the sole evidence necessary for diagnosis.

A positive result from a patient whose history and symptomatology are suggestive of amœbic disease, but whose stool examination gives negative results, is supportive evidence of disease. It indicates the necessity for further stool examination, or in appropriate circumstances helps to justify test therapy.

A positive result is strong supportive evidence of the diagnosis in a patient (with negative stool findings) whose sigmoidoscopic findings are suggestive.

A positive result in a patient who has undergone anti-amœbic treatment is strong evidence that the patient is not cured. (It is common experience among all observers that a positive response prior to effective treatment rapidly becomes negative.)

A negative result is not a reliable indication that the patient is not suffering from amœbic disease (whether acute, subacute or chronic).

Whether the patient has had or has not had antiamœbic treatment, a negative result which has previously been positive does not necessarily mean that the patient no longer harbours the organism. The test may suggest that the organism is not producing active disease.

There is a reasonably close coincidence between serological and sigmoidoscopic findings. This may have a significance from a serological viewpoint. We believe it has.

We would emphasize, in regard to the diagnosis of amœbiasis, by whatever means this is made, this guiding principle: it is one thing to diagnose the presence of *E. histolytica*; it is another thing to be satisfied that the infection is responsible for the patient's symptoms.

In conclusion, it can be admitted that it is possible, with the routine dose of complement used in order to avoid non-specific reactions, that the results from some sera with relatively smaller amounts of antibody may have been recorded as negative. It is possible that the commercial antigens may not have been able to react with every "positive" serum, and that they may require the addition of regional strains of *E. histolytica* to react with as many sera as possible from Australian ex-servicemen and ex-servicewomen or other people.

Acknowledgements.

The preparatory laboratory work on which this serum test was developed was carried out at the Prince Henry Hospital, Sydney, in the laboratory of Dr. B. Jones, and also with the help of the late Professor Albert Platt. I am indebted to Dr. Clive Backhouse for considerable help throughout the six years of the investigation, and to Mr. H. Clark, of Saint Vincent's Hospital. I am grateful to the members of the Repatriation Commission of the Commonwealth of Australia for the opportunity to take part in this work at their General Hospital at Concord, Sydney. There is a debt owing to the technical assistants and others of the pathology department, including Dr. T. Constance, who have each contributed something to this study over the years.

CARDIAC MURMURS IN SCHOOL CHILDREN.

By Douglas Stuckey,[1] Bryan Dowd and Helen Walsh.
From The Institute of Child Health, Royal Alexandra Hospital for Children, Sydney.

The present survey was carried out at the suggestion of Professor Lorimer Dods, Professor of Child Health in the University of Sydney, with the help and cooperation of Dr. E. S. A. Meyers, Director of the School Medical Service, and his staff, and with assistance from the staffs of the cardiac clinics at Saint Vincent's Hospital and the Royal North Shore Hospital. The purpose of the survey was to estimate the incidence of rheumatic and congenital heart disease among school children in Sydney.

Method.

The survey was carried out in the latter half of 1955. During the period of the survey the school medical officers referred for further investigation every child considered to have a cardiac murmur at routine medical examination. The total number of children examined in this time was 34,863, from 125 different schools in the Sydney metropolitan area. The two sexes were almost equally represented in this sample, and the ages ranged from five to sixteen years.

The total number of children reported to have a cardiac murmur was 467 (1·34%), and of these, 364 attended for further investigation at one of the special centres. Two hundred and twenty-eight children were examined at the Institute of Child Health and 136 at the other two centres. A careful history was taken, with particular attention to the presence or absence of symptoms, the age at which the murmur was first noted, and any illness suggestive of rheumatic fever. Physical examination was carried out by at least two observers with special cardiological experience. At the Institute of Child Health electrocardiographic and radiological examinations were carried out whenever the presence of an organic lesion was suspected; at the other centres these examinations were carried out as a routine procedure. Other special investigations, such as estimations of the hæmoglobin value and blood sedimentation rate, were carried out when indicated.

Diagnostic Criteria.

A diagnosis of rheumatic fever was made after all the available evidence had been referred to the criteria of Duckett Jones (1944). In six children, findings characteristic of rheumatic heart disease were noted in the absence of a history of rheumatic fever. In minimal mitral valve disease diagnosis rested on the presence of a soft pansystolic murmur, maximal at the mitral area, well heard laterally, not well propagated medially and inaudible at the aortic area, in the absence of other abnormal signs. Established mitral incompetence, aortic incompetence and mitral stenosis were recognized by the criteria laid down by Wood (1954).

A diagnosis of congenital heart disease was based on the criteria given by Wood (1950) and on experience at the congenital heart disease clinic, Royal Alexandra Hospital for Children, over a period of seven years (Stuckey, 1954). Of the 40 children with a congenital heart defect examined at the Institute of Child Health, 18 had been fully investigated at a special clinic, and in four others the diagnosis had been confirmed at operation.

The criteria of diagnosis of innocent systolic murmurs have been discussed elsewhere (Stuckey, 1955), and may be stated briefly as the presence of a mid-systolic murmur of short duration, of soft or moderate intensity and usually maximal in the left parasternal line, in the absence of any other abnormal findings on clinical, electrocardiographic and radiological examination.

[1] In receipt of a grant from the Rheumatic Fever Research Fund, University of Sydney.

Results.

The results are set out in Table I. The children examined at the Institute of Child Health and at the other centres are recorded separately, to show that independent groups of observers reached conclusions which did not differ very much from each other.

In Table II, those in whom no murmur was heard were excluded and the relative incidence of various forms of heart disease in school children with murmurs is shown.

It will be seen that of every 10 school children in whom a cardiac murmur was heard, seven were considered to have a normal heart with an innocent systolic murmur, two were thought to have congenital heart defects, and one was considered to be suffering from rheumatic heart disease.

The relative incidence of the various forms of heart disease in the 103 children with cardiac murmurs who did not attend for special examination may be assumed to be the same as in the 364 children who were so examined. If this assumption is made, the incidence figures for the 34,863 children included in the survey can be given as follows:

Rheumatic heart disease	1·4 per thousand.
Congenital heart disease	2·8 per thousand.
Innocent systolic murmurs	9·0 per thousand.

Some further details can be given about the children examined at the Institute of Child Health. Of 26 with rheumatic heart disease there were 12 with minimal mitral valve disease, 13 with mitral incompetence, three of whom had slight aortic incompetence as well, and one with fully established mitral stenosis. The average age at the time of examination was 11·2 years, and the sex incidence was equal. In only two cases had a murmur been noted before the age of five years. Twenty children had a past history of rheumatic fever, six did not.

TABLE I.

Incidence of Various Forms of Heart Disease.

Diagnosis.	Institute of Child Health.	Other Centres.	Total.
Rheumatic heart disease ..	26	10	36
Congenital heart disease ..	40	32	72
Innocent systolic murmur ..	145	94	239
Normal heart, no murmur ..	17	—	17
Total	228	136	364

Forty children were considered to have congenital heart defects, and the numbers with particular lesions were as follows:

Ventricular septal defect	13
Patent ductus arteriosus	5
Atrial septal defect	7
Pulmonary stenosis	8
Aortic stenosis	3
Coarctation of the aorta	1
Tetralogy of Fallot	2
Eisenmenger's complex	1

The average age at the time of examination was 9·5 years, and females predominated in the proportion of 26 to 14. In 23 instances a murmur had been noted before the age of five years.

There were 145 children considered to have innocent systolic murmurs. Their average age was 9·2 years, and in 17 instances a murmur had been noted before the age of five years. In 41 the murmur was considered to be of pulmonary origin, and females predominated in the proportion of 31 to 10. In 96 the murmur was regarded as of aortic origin, and males predominated in the proportion of 54 to 42. In 18 children the murmur was maximal in the third and fourth left intercostal spaces near the sternum and not heard elsewhere, and a decision could not be reached as to whether it was of pulmonary or aortic origin. The innocent murmurs of aortic origin are discussed more fully elsewhere (Stuckey, 1956).

TABLE II.

Percentage Incidence of Various Forms of Heart Disease.

Diagnosis.	Institute of Child Health.	Other Centres.	Total.
Rheumatic heart disease ..	12·3	7·4	10·3
Congenital heart disease ..	19·0	23·2	20·7
Innocent systolic murmur ..	68·7	70·4	69·0

Discussion.

The incidence of all forms of organic heart disease in school children in this survey (4·2 per thousand) is much the same as previous figures reported in Australia. For instance, Graham (1937) found that the incidence of "organic hearts" in Victorian school children over a period of seven years up to 1936 was 4·32 per thousand, and Maddox (1937) quoted similar figures for New South Wales for the years 1929 to 1934 ranging from 3·2 to 4·4 per thousand. Some American figures for similar types of school surveys are 5·0 per thousand in New York (Halsey, 1921), 3·6 per thousand in San Francisco (Sampson *et alii*, 1938), 5·3 per thousand in Cincinatti (Rauh, 1939), 4·4 per thousand in San Francisco (Robinson *et alii*, 1948) and 3·6 per thousand in Toronto (Keith, 1951). It must be remembered that these figures do not indicate the total incidence of organic heart disease in childhood, for no account is taken of children being treated in hospitals or at home and unable to attend school.

In this survey congenital heart defects accounted for no less than two-thirds of all cases of organic heart disease in school children. For comparison, figures from similar surveys in America are 57·9% in San Francisco (Sampson *et alii*, 1938), 45% in Cincinatti (Rauh, 1939) and 56% in Toronto (Keith, 1951). Some English figures are 54% (Price, 1949) and 41% (Appley and Perry, 1954). Earlier surveys gave a much lower proportion of congenital heart disease, some as low as 14% (Halsey, 1921). It is a matter for speculation whether the higher proportions of congenital heart disease now reported are due to improved diagnostic accuracy, to an increased incidence of congenital heart defects, or to a smaller incidence of rheumatic heart disease in recent years.

The figures in this report may be of help to the practitioner faced with the common problem of the significance of a cardiac murmur discovered incidentally during the examination of an otherwise healthy child.

Summary.

In a survey of 34,863 school children in the Sydney metropolitan area, the incidence of rheumatic heart disease was 1·4 per thousand and that of congenital heart disease 2·8 per thousand.

Of every 10 children found to have cardiac murmurs, one suffered from rheumatic heart disease, two had congenital heart defects and the remaining seven were considered to have normal hearts with innocent systolic murmurs.

Acknowledgements.

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INNOCENT SYSTOLIC MURMURS OF AORTIC ORIGIN.

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IN a previous paper (Stuckey, 1955) a series of children with innocent systolic murmurs was reviewed, and it was suggested that these murmurs were due to variations in the shape or calibre of the great vessels distal to a normal pulmonary or aortic valve. In a recent survey of cardiac murmurs in school children reported elsewhere (Stuckey *et alii*, 1956), the opportunity arose to examine a further large number of children considered to have innocent systolic murmurs, and the present report is based on this material.

Of 228 school children with cardiac murmurs investigated at the Institute of Child Health, Royal Alexandra Hospital for Children, 145 were considered to have innocent systolic murmurs, and of these murmurs, 96 were regarded as being of aortic origin and will be discussed in more detail. The murmurs of aortic origin were short, mid-systolic and of soft or moderate intensity. The murmur was maximal in 79 instances in the third or fourth left intercostal space close to the sternum, in seven at the mitral area, and in 10 at the aortic area. It was characteristic that these murmurs were heard widely over the precordium, in particular at both aortic and mitral areas and at intermediate points between them. In 40 children the murmur was described as squeaky or musical. Males predominated in the proportion of 54 to 42. No other abnormal signs were found on clinical, radiological or electrocardiographic examination.

Discussion.

Innocent systolic murmurs of aortic origin are not well known, and yet this survey suggests that they are at least as common in normal healthy children as those of pulmonary origin. These murmurs were referred to by Wedum and Rhodes (1955) as musical or "fiddle-string" murmurs, and as soft, short, systolic murmurs heard between the cardiac apex and the sternum or at the aortic area; but no suggestion was offered as to their origin.

The evidence in favour of aortic origin rests on the character and distribution of the murmurs. They are strictly mid-systolic in timing, corresponding to the phase of maximum ejection of blood from the ventricles into the great vessels; and they are heard along the line of the left ventricular outflow tract from the cardiac apex to the aortic area and on into the great vessels of the neck, and may be maximal anywhere along this line. In these two characteristics the murmurs closely resemble those of aortic stenosis, which, however, are longer, louder and usually accompanied by a thrill. In addition, patients with aortic stenosis usually show some evidence of left ventricular hypertrophy either clinically or in the electrocardiogram, and in more severe cases the heart is enlarged on radiological examination and characteristic changes occur in the peripheral pulses and in the blood pressure.

The most likely explanation of innocent systolic murmurs of aortic origin is a slight disproportion between the size of the normal aortic valve orifice and the calibre of the ascending aorta beyond, sufficient to produce eddying, turbulence and cross-currents and so an audible murmur. This explanation is probably not capable of proof, for the opportunity of post-mortem examination seldom occurs, and when it does the measurements may well be within normal limits of variation. It is conceivable that such a disproportion may occur transiently during childhood when different structures grow at different rates, and this may explain why these murmurs are comparatively common in the child and uncommon in the adult.

The high-pitched squeaky or musical character of many of these innocent murmurs is important as a diagnostic point. This was noted in almost 40% of those considered to be of aortic origin in this survey, and in none of those of pulmonary origin. Organic murmurs of aortic origin are usually low-pitched and harsh, and a musical character is not common.

Innocent systolic murmurs of aortic origin cause diagnostic difficulties much more frequently than those of pulmonary origin, principally because the aortic murmurs are well heard and sometimes maximal at the mitral area. The distinction from aortic stenosis has already been mentioned. The systolic murmur of ventricular septal defect is louder and longer, usually accompanied by a thrill, and often very localized when the defect is small: larger defects show other abnormalities on examination. The commonest diagnostic difficulty lies in the distinction from the mitral systolic murmurs of rheumatic heart disease. These murmurs, while variable in intensity, are true pansystolic murmurs and extend throughout systole, and are best heard at and lateral to the mitral area. It can be taken as a safe working rule that a systolic murmur originating at the mitral valve cannot be heard at the aortic area even when the murmur is loud, and this is the most useful distinction from innocent systolic murmurs of aortic origin.

It is important to realize that in a child suspected of having rheumatic fever, the mere presence of a systolic murmur at the mitral area is not sufficient to support the diagnosis; such a murmur must have the characteristics of a murmur originating at the mitral valve. If the murmur is soft, short, maximal at the left border of the sternum, and audible at the pulmonary or aortic areas, it is almost certainly an innocent systolic murmur, and cannot be regarded as evidence of rheumatic carditis.

Summary.

Innocent systolic murmurs of aortic origin are at least as common as innocent pulmonary systolic murmurs in normal healthy children.

Their characteristics are discussed in detail, and an explanation is offered for their origin.

The importance of distinguishing these murmurs from those due to congenital and rheumatic heart disease is stressed.

Acknowledgements.

Thanks are due to Dr. Bryan Dowd and Dr. Helen Walsh for their assistance in the examination of these patients.

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DROWNING.

By KEITH BOWDEN,
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Is our understanding of the cause of death in drowning and its treatment adequate?

From time to time, in examining the bodies of drowned persons, one meets with cases in which the usual signs or appearances of death from drowning are absent. If it was not for the known facts that the body had been recovered from water *et cetera*, one would not be able to determine from macroscopic inspection of the organs alone that death was due to drowning, and in many cases the most that one can say at the conclusion of the autopsy is that the appearances are consistent with death from drowning.

Because of these difficulties a search has gone on for some post-mortem finding that is diagnostic of death from drowning. In other words, the absence of reliable evidence of drowning at autopsy has led to a search for some specific test.

It has been known for many years that water may be absorbed from the lungs into the blood-stream. Various investigators have demonstrated this in animals. For example, Colin introduced 25 litres of water into the trachea of a horse over a period of six hours without significant interference with pulmonary ventilation; six kilograms of blood were withdrawn to offset any hydræmia (1873; see Moritz, 1944).

When it was realized that water might enter the blood-stream by way of the lung alveoli and might quickly reach the left side of the heart, attention was focused, in fresh-water drowning, on the possible dilution of the substances in the blood on the left side of the heart, as compared with the blood in the right side of the heart, as an aid in the diagnosis of death from drowning. Examination has been made of the level of the various constituents in the blood for evidence of the entry of water or electrolytes into the blood-stream.

Gettler (1921) examined the chloride level in the blood in cases of drowning, and found that in salt-water drowning the chloride level in the blood on the left side of the heart was elevated and was higher than the corresponding finding in the blood on the right side of the heart. In fresh-water drowning the chloride level in the blood on the left side of the heart was lower, and below that in the blood on the right side of the heart. He concluded that a difference of more than 25 milligrammes between the chloride levels on the left and right sides of the heart indicated that death was due to drowning—so there came into existence "Gettler's test".[1] This was an important contribution.

Subsequent investigation agrees in part with Gettler's conclusions; but a difference of very much more than 25 milligrammes may exist in the chloride levels in blood taken from the left and the right sides of the heart in cases in which death is not due to drowning. There may be a difference up to 50 milligrammes in the chloride level between the two sides of the heart when death is due to natural causes. There are very definite limitations to Gettler's test. It is not specific, and in many cases of drowning, if an appreciable interval has elapsed since death (delayed drowning) it is not of much assistance to the pathologist.

Examination has been made in drowning of the other constituents of the blood, estimation of freezing point, specific gravity, *et cetera*, in an attempt to determine whether there is any difference between the blood on the left side of the heart and that on the right side of the heart; but none of these estimations gives a specific test for drowning.[2] It has been stated that an estimation of the iron content of the blood appears to be as satisfactory as that of any of the other constituents. After death putrefaction, hæmolysis, autolysis, diffusion, *et cetera*, may all alter the composition of the blood.

Jetter and Moritz (1943), in investigating this problem, threw valuable light on it. They showed that various changes occurred, for example, in the chloride level of the blood after death. In general, after death the chloride level falls. It falls on both sides of the heart, but it does not fall in any constant fashion. The fall in the chloride level in the blood of the heart after death may be more pronounced on one side of the heart in one case, more pronounced in the blood on the other side of the heart in another case. The magnesium level in the blood tends to rise *post mortem*, and the level of magnesium, which has also been used as a diagnostic test in drowning, is also subject to post-mortem variation.

One important conclusion to be drawn from the work of Jetter and Moritz is that it is first essential to determine just what happens to the various constituents of the blood after death in ordinary cases in which death is not due to drowning. They concluded that within six to twelve hours after death there was a progressive fall in the blood chloride level and a rise in the magnesium level. This has to be clearly kept in mind when one is interpreting post-mortem analysis of the blood constituents in cases of suspected drowning, and again it imposes limitations on their use. This is most obvious when, for example, the significance of potassium levels is being considered in analysis of post-mortem blood. Insufficient work has been done on this aspect of the subject.

Swann and his co-workers made observations on drowning in anæsthetized dogs (1949, 1951). They showed that in fresh-water drowning there was a rapid dilution of the blood with water gaining entrance from the alveoli, and that rapidly ventricular fibrillation ensued. Analysis of the fluid in the bronchial tree also suggested that pulmonary œdema developed during drowning. On the other hand, in salt-water drowning, water appeared to pass out from the blood-stream into the alveoli, and electrolytes passed into the blood-stream from the fluid in the alveoli, thus bringing about a greater concentration of these substances such as chloride and magnesium in the blood. In salt-water drowning in the dogs examined ventricular fibrillation did not occur, and again analysis of the fluid in the bronchial tree suggested that pulmonary œdema developed.

Observations on animals raise some interesting questions in man. For example, is death in fresh-water drowning due to ventricular fibrillation, brought about by the rapid flooding of the blood-stream with water? Does the mechanism of death differ in fresh-water drowning from salt-water drowning, in which ventricular fibrillation has not been so commonly observed? Could the rise in magnesium level in the blood in salt-water drowning of itself lead to death? In other words, are there additional factors, changes in the blood, apart from the fundamental one of asphyxia or anoxia—additional factors such as ventricular fibrillation in fresh-water drowning, alteration in osmotic pressure, alteration in the electrolyte levels, *et cetera*, themselves sufficient to bring about disastrous results? Are these alterations sufficient in themselves to be incompatible with life? Is death in drowning a much more complex problem than simple asphyxia, opening up other possibilities in treatment and the resuscitation of the apparently drowned?

If in the man who has been apparently drowned in fresh water, ventricular fibrillation has rapidly occurred, is artificial respiration sufficient treatment, or is artificial respiration merely being applied to a man who is already dead or dying from the effects of the changes in the composition of his blood? In other words, as well as asphyxia, is this man dying from cardiac arrest or ventricular fibrillation or medullary failure due to blood which is so altered as to be incompatible with life when it enters the coronary arteries or reaches the medulla? If this is so, is there a place for additional treatment, such as cardiac massage, the use of defibrillator, or some form of intravenous therapy?

In posing these questions the following evidence is brought forward. An analysis was made of the blood

[1] Whole blood was used.

[2] For a review of this subject see Moritz, 1944.

constituents indicated in Tables I to IV in cases of drowning in man, both in fresh water and in salt water. To eliminate as far as possible post-mortem changes, no cases were included in this series in which the blood was not obtained within four hours of the drowning event.

Analysis of sea-water gives the following electrolyte levels in milliequivalents per litre: sodium, 440; magnesium, 101; calcium, 18; potassium, 10; chloride, 510.

TABLE I.

Human Beings: Drowned Less than Four Hours Previously in Fresh Water.

Subjects.	Serum Electrolyte Levels (Milliequivalents per Litre).			
	Sodium.	Potassium.	Magnesium.	Chloride.
Normals	140 to 150	3·5 to 5·0	1·0	100 to 110
Case I:				
Right side of heart ..	141	22·0	—	91·0
Left side of heart ..	116	20·0	—	76·0
Tracheal fluid	83	13·0	—	72·0
Case II:				
Right side of heart ..	129	25·0	2·9	91·2
Left side of heart ..	129	22·5	2·8	95·0
Case III:				
Right side of heart ..	138	6·6	2·25	94·0
Left side of heart ..	120	8·2	2·42	84·0
Tracheal fluid	71	16·3	0·9	61·0
Case IV:				
Right side of heart ..	130	10·0	3·7	92·0
Left side of heart ..	133	5·6	3·3	93·0
Case V:				
Right side of heart ..	151	7·2	2·9	108·9
Left side of heart ..	151	5·3	1·5	107·8
Case VI:				
Right side of heart ..	145·5	19·8	2·6	—
Left side of heart ..	146·0	18·7	2·5	—

Analysis of Findings.

Drowning in Fresh Water.

CASE I.—There is a significant fall in the sodium level on the left side of the heart, with a pronounced fall in the chloride level on the left side of the heart and a pronounced rise in the potassium level on both sides of the heart. Apart from the high potassium level, the change is similar to that reported in the experimental animal in fresh-water drowning when the analysis has been made on serum immediately after death. The potassium level is very much higher than that reported in the experimental animal, and reference to the control series of cases shows that this high potassium level could be due to post-mortem change. There is evidence here of dilution with water of the blood on the left side of the heart. The findings also suggest that death was rapid, as there is no fall in the sodium content of the blood in the right side of the heart, and only a slight fall in the blood chloride content in the right side of the heart. This would be consistent with rapid death from cardiac arrest or ventricular fibrillation before the blood on the left side of the heart had time for more than one complete circuit. Clinically, human beings with sodium levels as low as this have been comatose when suffering from water intoxication. Although in the control series of cases the sodium level has dropped significantly in one case (Case II, Table IV), the sodium content has dropped significantly on both sides of the heart within four hours of death. Clearly the change in potassium level during the drowning process cannot be evaluated satisfactorily in this case. It could not be claimed from these results that the change in potassium, either in the rate of change or in the amount, was significant in the death, because it is obviously not possible to say how much of this is of post-mortem origin.

These findings do not negate the suggestion that a rapid rise in serum potassium level may be an important factor in bringing about cardiac failure in fresh-water drowning. These cases simply show that in human beings, in the interpretation of the potassium level of the serum in cases of drowning, due allowance must be made for changes after death. The difficulty lies in the inability of the investigator to obtain serum sufficiently soon after death as to be able to discount post-mortem change. There was insufficient serum in Case I for magnesium estimation.

CASE II.—The blood sodium level in each side of the heart is lowered, as is the chloride level. The potassium level is again very high, and there is some rise in the magnesium level. The rise in magnesium, like the rise in potassium, can be explained on the basis of post-mortem change. The fall in both chloride and sodium levels on both sides of the heart suggests that there was hæmodilution, and that there may have been more than one circuit of the diluted blood. In the light of Case II, Table IV, of the controls, it is theoretically possible that all the changes observed in Case II of fresh-water drowning could possibly be of post-mortem origin.

TABLE II.

Human Beings: Salt-Water Drownings.

Subjects.	Serum Electrolyte Levels (Milliequivalents per Litre).			
	Sodium.	Potassium.	Magnesium.	Chloride.
Case VII:				
Right side of heart ..	154·0	15·5	4·5	115·6
Left side of heart ..	156·0	14·5	8·5	119·7
Case VIII:				
Right side of heart ..	161·5	22·6	10·5	120·0
Left side of heart ..	184·0	13·1	20·5	151·2
Case IX:				
Right side of heart ..	159·0	22·0	3·8	—
Left side of heart ..	172·0	19·0	7·4	—
Case X:				
Right side of heart ..	162·0	21·2	3·7	124·0
Left side of heart ..	172·0	13·5	3·7	146·0
Case XI:				
Right side of heart ..	179·0	16·1	5·3	—
Left side of heart ..	192·0	14·0	9·5	—

CASE III.—The findings in Case III are also similar to those in the reported cases of fresh-water drowning in animals, and these findings could again mean hæmodilution on the left side of the heart and rapid death similar to that in Case I. The potassium findings are also in keeping with those found in the experimental animal.

CASE IV.—In Case IV there is a fall in the sodium level on both sides of the heart, a fall in the chloride level on both sides of the heart, a more pronounced rise in the potassium level on the right side of the heart, and some elevation of the magnesium level on both sides of the heart. These findings again could mean that there has been hæmodilution with more than one circuit of the blood, or possibly all these changes could be of post-mortem origin.

CASE V.—In this case there is no significant change and nothing to suggest hæmodilution.

CASE VI.—In this case there is no significant change in the sodium level; there is a high level of potassium. There was insufficient serum for further analysis. The change in potassium level in this case can be accounted for on a post-mortem basis.

A feature in fresh-water drowning in the reported experiments on animals is the rapid hæmolysis occurring in the blood, especially on the left side of the heart. In all the cases of fresh-water drowning in human beings examined here, hæmolysis was a feature. From a consideration of the reported work on animals and the frequency of ventricular fibrillation in fresh-water drowning, it is tempting to suggest that hæmolysis with rapid liberation of potassium from the cells can be a significant factor. A high potassium level such as is found in these cases of drowning in human beings could be accounted for by post-mortem hæmolysis. Potassium is much more abundant in the cells than it is in the plasma, and with the hæmolysis that occurs after death (control series) one would expect a rise in serum potassium level after death. In addition to an agonal release of potassium, the potassium content of the serum greatly increases on standing in contact with the red blood corpuscles.

What has actually happened to the serum potassium level in these cases of drowning in human subjects is masked by what is obviously post-mortem change, and so in these series of cases this problem is not satisfactorily answered.

Rath ("Current Comment", 1954) reported the case of a boy, aged fifteen years, in whom hæmoglobinæmia and hæmoglobinuria were observed during his recovery from submersion. The boy was found lying in a swimming pool,

TABLE III.
Control Series.

Subjects.	Serum Electrolyte Levels (Milliequivalents per Litre).			
	Sodium.	Potassium.	Magnesium.	Chloride.
Hanging.				
Case I: 24 to 36 hours after death:				
Right side of heart ..	121·0	5·3	3·0	82·0
Left side of heart ..	118·0	6·3	3·2	87·0
Case II: About 6 hours after death:				
Right side of heart ..	158·0	14·0	1·64	—
Left side of heart ..	160·0	11·4	2·9	—
Case III: About 12 hours after death:				
Right side of heart ..	—	—	3·08	110·0
Left side of heart ..	—	—	1·18	110·0
Case IV: About 8 hours after death:				
Right side of heart ..	144·5	18·0	3·6	98·3
Left side of heart ..	140·5	18·5	3·4	101·3
Case V: About 5 hours after death:				
Right side of heart ..	118·0	35·5	3·0	89·4
Left side of heart ..	143·8	24·0	2·5	106·3
Head Injury.				
Case VI: About 4 hours after death:				
Right side of heart ..	139·0	19·5	4·1	104·6
Left side of heart ..	146·0	19·0	4·6	115·3

having been there for an unknown period, thought to be not more than five minutes. Voluntary respiration was resumed at once when artificial respiration was applied, and oxygen was given while he was being removed to hospital. Here he was restless, exhaling sanious fluid, and scattered rales were audible in the lungs. Dark urine was passed which contained protein, some red cells and many white cells, and tests showed the presence of blood pigment. "Sodium Amytal" was given to facilitate a lumbar puncture. The boy lost the usual evidence of reflex activity and showed signs of anoxæmia. These signs subsided, and during this period signs of decerebrate rigidity were manifest; but on the following day the boy had regained memory and ordered speech. After this he made a rapid recovery. The investigation carried out on this boy pointed to the cause of the hæmoglobinæmia as intravascular hæmolysis produced by the absorption of hypotonic fluid—water—into the circulation.

Salt-Water Drowning.

CASE VII.—In this case the sodium level as well as the chloride level is elevated a little on each side of the heart. There is a pronounced rise in the potassium level on both sides of the heart. The magnesium level is raised on both sides of the heart, but much more so on the left side than on the right. These findings are consistent with some hæmoconcentration and with the passage of electrolytes from sea water into the blood.

CASE VIII.—There is a pronounced rise in the sodium level on the left side of the heart, a pronounced rise in the chloride level on the left side of the heart, and a pronounced rise in the magnesium level on the left side of the heart; again, the potassium level is elevated on both sides.

CASE IX.—The sodium level is much raised on the left side of the heart, the magnesium level is high on the left side of the heart, the potassium level is raised on both sides. There was insufficient serum for chloride estimation.

CASE X.—The sodium and the chloride levels are raised, but much more so on the left side of the heart than on the right. There is a pronounced elevation in potassium level with some rise in the magnesium level.

CASE XI.—The sodium level on the left side of the heart is very high. The magnesium level is raised, more so on the left side of the heart, and the potassium level is elevated on each side.

In general, in these cases of salt-water drowning there is an increase in the electrolyte levels, more pronounced on the left side of the heart than on the right, although there is still an increase on both sides. This is also in agreement with the reported findings in the experimental animal when the analysis has been made immediately after death; but again the same objections in interpretation apply to potassium. This pronounced rise in potassium level again is explicable on a post-mortem basis.

TABLE IV.
Sudden Death Less than Four Hours Previously.

Subjects.	Serum Potassium and Sodium Levels (Milliequivalents per Litre).	
	Potassium.	Sodium.
Case I:		
Left side of heart ..	17·2	137·0
Right side of heart	26·8	134·0
Case II:		
Left side of heart ..	48·6	91·0
Right side of heart	46·9	103·0
Case III:		
Left side of heart ..	13·3	143·0
Right side of heart	13·3	146·0

These findings indicate the rapid passage, for example, of chloride and magnesium into the blood passing through the left side of the heart.

In one series of experiments on the dog (Trethewie, unpublished work), ventricular fibrillation and tachycardia occurred. The high magnesium level, especially in the serum on the left side of the heart, raises the question whether cardiac and respiratory arrest could be due to this change. A level of 6·0 to 14 milliequivalents of magnesium per litre is considered as the upper limit compatible with life. There is such disproportion between the magnesium and calcium contents of sea-water that this rapid absorption of magnesium and high level in the serum could be an important factor.

Post-Mortem Considerations.

In these two series of cases—that is, fresh-water drowning and salt-water drowning—the changes in the electrolyte levels tend to be in opposite directions. For example, the elevation of both sodium and chloride levels in salt-water drowning is not what one expects to find as a result of post-mortem change, and is in the opposite direction to the changes in sodium and chloride levels, for example, in fresh-water drowning. This tendency to a change in opposite directions also indicates that asphyxia alone is not a common factor setting up the alteration.

Biochemical Tests for Drowning.

Provided that their limitation is appreciated, biochemical tests may be of value as an aid in the diagnosis of drowning. This is seen, for example, in the fall in the sodium level on the left side of the heart in Cases I and II; in the fall in the chloride level on the left side of the heart in Cases I and III (Gettler's test); in the high rise in chloride level on the left side of the heart in Cases VIII and X (Gettler's test); in the high magnesium level on the

left side of the heart in Cases VII, VIII, IX and XI. In fact, there is ample biochemical evidence of drowning in all the cases of salt-water drowning studied here.

Pulmonary Œdema.

One of the typical findings in cases of drowning both in fresh and in salt water is the presence of a whitish frothy fluid, sometimes blood-stained, in the bronchial tree and in the lungs. There may be a great amount of this, or there may be practically none—the lungs are dry. The presence of frothy fluid filling the air passages has always seemed to be a formidable barrier to the entry of air during artificial respiration, unless some attempt is made to get rid of it either by making as much use of gravity as possible in artificial resuscitation, or by sucking out the air passages mechanically. What is its origin? We were brought up as students in the belief that the entry of water into the air passages, together with the efforts at respiration during drowning, led to the churning up of a mixture of water, air and mucus from the bronchial tree, with the production of this whitish froth. Chemical analysis of the fluid removed from dogs in Swann's experimental work indicated that active pulmonary œdema could be taking place. This has been simply and strikingly shown by one observer in the dog and in the cat. These animals were removed from water before death occurred and before the animals had ceased to breathe. Although there was no frothy fluid in the air passages on withdrawal from the water, suddenly in some of the animals whitish frothy fluid in large amounts began to well out from the nostrils and the mouth. It was apparently the onset of acute pulmonary œdema. In some animals it did not develop—the air passages remained dry. This parallels the findings in drowning in human subjects. Some have an apparently dry bronchial tree. Others show filling of the air passages with whitish frothy fluid, sometimes blood-stained, the lungs are heavy, and the alveoli contain this fluid.

The onset of active acute pulmonary œdema late in the drowning process satisfactorily explains the various autopsy findings. In some cases there is no fluid in the lungs—evidently death occurs before the point is reached at which acute pulmonary œdema sets in. In others there is striking pulmonary œdema—this has set in late in the drowning process before death occurs. In those cases of so-called delayed drowning in which the victim has been rescued or has reached safety only to succumb afterwards for no obvious reason, pulmonary œdema has been the common finding. Various explanations could be put forward to account for the onset of acute pulmonary œdema or its absence; but whatever the mechanism, it appears to be an active complicating event during the drowning process.

Ventricular Fibrillation.

Experimental work indicates that in the dog, in cases of fresh-water drowning, ventricular fibrillation sets in rapidly, and that this is probably the underlying important factor in the death of these animals. In Trethewie's series of experiments in the dog, ventricular fibrillation set in in the cases of fresh-water drowning. It also occurred in one of three dogs drowned in salt water, in the second dog it set in when the chest was opened after cessation of respiration, and in the third dog drowned in salt water there was great tachycardia.

In the human subject biochemical analysis shows that the changes in some cases studied parallel the findings in the animal work which has been recorded.

It is probable, then, that in some cases of fresh-water drowning, and in some cases of salt-water drowning, cardiac arrest or ventricular fibrillation may rapidly set in and may be the disastrous complicating factor as a result of the biochemical changes in the blood.

Cessation of Respiration.

Although in drowning respiration ceases before the electrical activity of the heart, as shown by electrocardiographic studies, nevertheless the heart's activity as recorded in the dog by the electrocardiograph is frequently so feeble during this period that some form of resuscitative treatment applied to the heart would seem desirable when the question of resuscitation is considered. In one series of cases it was noticeable that when the heart had ceased to beat and when the chest was opened, there was pronounced distension of the pericardial sac, and the opening of the sac and the handling of the surface of the heart were sometimes followed by restoration of the heart beat.

In drowning in the animal in the experiments studied and recorded, respiration ceased before activity of the heart ceased. This has been the accepted teaching in the case of the human being in drowning, and it has been the basis of treatment. It has been considered that if the heart is still beating although respiration has ceased (as in electrocution, for example), artificial respiration may be sufficient to bring about recovery. As has previously been mentioned, electrocardiographic studies have shown in the dog that although respiration has ceased, the degree of muscular activity which persists after cessation of respiration, sometimes for a comparatively long period, is frequently extremely feeble, and it is doubtful, if this is the case in the human subject, whether artificial respiration of itself would be sufficient to bring about recovery.

Hæmolysis of the Blood.

Hæmolysis of the blood is a pronounced feature in cases of fresh-water drowning, not only in the animal studies but in the cases of fresh-water drowning studied in this paper. Some hæmolysis was also present, more pronounced on the left side of the heart than on the right side, in some of the cases of salt-water drowning. Again, post-mortem hæmolysis must be considered. That it occurs in the human subject in life is borne out by the study of the case previously quoted—that reported by Rath—so that potassium liberation could be important. Clinical observations on similar subjects admitted to hospital, who recover, would be worth while in this regard.

Treatment.

Is it necessary in the treatment of the apparently drowned to do more than attempt to clear the airway and maintain artificial respiration either manually or mechanically? If necessary, is it practicable?

The biochemical studies in cases of drowning indicate that there are pronounced changes in the blood accompanying the drowning process. These changes can be so great as by themselves to render recovery extremely unlikely unless some treatment is directed toward them. If human beings drowned in fresh water have rapidly succumbed to anoxia and ventricular fibrillation, or to cardiac arrest, then it appears that artificial respiration is insufficient to bring about recovery. Is there a place for cardiac massage? Should cardiac massage be attempted almost at once, if the subject taken from the water soon after immersion shows no signs of respiration or of life? Is there a place for the use of a defibrillator in such cases? If these biochemical changes are occurring, is there a place for venesection or some form of corrective intravenous treatment? Is it necessary, for example, in fresh-water drowning, to give some form of intravenous therapy? Would this be of value—the intravenous injection, for example, of calcium solution or hypertonic saline? On the other hand, in cases of salt-water drowning, is it necessary to attempt to dilute the blood with the intravenous injection of water or hypotonic saline?

Conclusion.

Experimental work and these studies indicate that drowning is a very much more complicated process in many instances than simply deprivation of air, or anoxia; but this still remains the fundamental problem, and the treatment must still fundamentally and initially remain the same. Is it necessary to add to the already accepted treatment along the lines that have already been indicated?

Of course, additional treatment along these lines in some instances appears to be largely impracticable, but it is not necessarily so.

If, then, drowning is the complicated process that has been suggested, then it becomes all the more important—because it appears to be all the more difficult to treat successfully a subject who has been apparently drowned—that learning to swim should be a fundamental part of every child's education.

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Reviews.

Surgery for General Practice. By Victor Richards, M.D.; 1956. St. Louis: The C. V. Mosby Company. Melbourne: W. Ramsay (Surgical), Limited. 10" × 7", pp. 947, with illustrations. Price: £9 12s. 6d.

THIS book is a product of the Stanford University School of Medicine and is written for the average physician in general practice. It is not detailed enough for the surgical specialist. No reference list is appended, and rarely is reference made to an individual's name or work. This book does simplify and clarify a vast store of surgical information, and is especially intended for those who are not in want of additional facts but require a better organization of facts already known.

A refreshing arrangement of subjects is presented. However, an excellently written chapter of 63 pages on the ear, nose and throat, one page on carcinoma of the breast, seven pages devoted to acute abdominal disorders, and only a passing reference to the subject of hernia give an unbalanced impression, especially if the book is to be recommended to students. More emphasis could be laid on the loss of sensitivity by organisms to antibiotics and the reemergence of acute osteomyelitis, acute empyema and acute mastoiditis as conditions again demanding earlier and more adequate surgery than has been the custom over the last decade.

For practitioners needing a rapid brush-up in many aspects of surgery today, this book can be recommended.

A Short History of Public Health. By C. Fraser Brockington, M.A., M.D., D.P.H., B.Chir. (Cantab.), M.Sc. (Manchester); 1956. London: J. and A. Churchill, Limited. 7½" × 5", pp. 243. Price: 15s.

BROCKINGTON has written a pleasant and useful little book on the history of his subject.

What is called "modern" public health began in the eighteenth century, when industrial progress also began. Early features were Frank's "medical policy", and the work of Percival, the general practitioner who studied the effects of industrial life on the dwellers in the new towns. From then the developments are succinctly described.

The book is in two parts. The first section traces the growth in the last two centuries of interest in community health. Bad sanitation and squalor, cholera epidemics and factory hazards were compelling problems. The call for care of women and children became insistent. The work of Chadwick, Southwood Smith, Shaftesbury and others was ardent and unceasing. The second portion of the book is a more detailed account of the development of health services, maternal and child care, infectious disease control, and care of the aged.

The development of social medicine is described thus: "The social approach must bring public health into closer touch with clinical medicine. The barriers which have separated them must be broken down." And, of the future, Brockington writes: "In pursuing the social and epidemiological approach, a new field of opportunity opens before the medical officer of health. As he allies himself with hospitals, general practitioners, industry and schools, he is in a position to bring the benefit of his social knowledge to the lives and homes of the people."

This book is designed especially to assist doctors, nurses and social workers doing diploma courses. For the general reader, medical or lay, it presents a fascinating account of how a great branch of medicine has grown.

The Treatment of Fractures. By Lorenz Böhler, M.D.; Volume I; 1956. New York and London: Grune and Stratton, Incorporated. 9½" × 7", pp. 1104, with 1721 illustrations. Price: $6.50.

TWO DECADES have passed since the last edition of Böhler's work on the treatment of fractures was published in English. During that time there has been another world war, in which Böhler was a consulting surgeon to the German armies. The 1914-1918 war gave Böhler a unique experience in war surgery. This was followed by his control of an accident hospital in Vienna. It is probably true to say that no surgeon living has had a greater experience in war and accident surgery. His teaching undoubtedly revolutionized the treatment of fractures in the early "thirties" in Great Britain, particularly by the introduction of the non-padded plaster cast as a method of splintage. Vienna at that time was the Mecca of all interested in traumatic surgery. Böhler's views must command respect.

The chapter on general principles of fracture treatment and wound management is particularly good. Böhler stresses repeatedly the importance of thorough wound toilet and ruthless excision of all devitalized tissue as the basis of all wound management. This lesson, learnt by bitter experience in the first war by British surgeons, had to be relearnt in the recent war.

The dangers of distraction are repeatedly stressed. In fact Böhler states: "One must aim at obtaining shortening of 1-10 mms. after any fracture, but by no means an elongation. This may be surprising, as we have up to now attempted to avoid any shortening."

The chapter on fractures and dislocations of the spine is good. Böhler continues to stress the importance of anatomical reduction where possible. The management of the paraplegics is well worth study.

Fractures of the upper limbs are dealt with in sequence. It is pleasing to find a detailed description of the management of that unusual combination of injuries—the fracture-dislocation of the shoulder. The difficult supracondylar fracture is dealt with by skeletal traction through the olecranon after preliminary reduction. One strange omission is the operative treatment for vascular damage or spasm in this type of fracture.

There are some excellent general rules and principles for treating gun-shot wounds of the upper limbs. This is an entirely new departure in any text-book and of great value for those who have had no experience in this type of surgery. Hand injuries are also ably dealt with and the methods of Bunnell advocated.

The book is well produced and (especially important) profusely illustrated. Altogether it is an outstanding work, which should be read by every surgeon whose work lies in the sphere of traumatic surgery.

Subarachnoid Hæmorrhage. By John N. Walton, M.D. (Durham), M.R.C.P.; foreword by Sir Charles Symonds, K.B.E., C.B., D.M., F.R.C.P.; 1956. Edinburgh and London: E. and S. Livingstone, Limited. 8½" × 6", pp. 366, with illustrations. Price: 30s.

BY the development of cerebral angiography into a relatively safe diagnostic procedure academic interest in

subarachnoid hæmorrhage has been converted into hope that a surgical attack upon intracranial aneurysms and angiomata may substantially lower mortality. Most recent literature on this important subject has emanated from radiological and neurosurgical clinics, and it is therefore of interest that what probably will become a standard reference book on subarachnoid hæmorrhage should be written by a physician. Walton's monograph is based on information obtained from a study of 312 cases of spontaneous subarachnoid hæmorrhage seen in hospitals at Newcastle-on-Tyne, in conjunction with an exhaustive review of the literature. The ætiology of subarachnoid hæmorrhage and the symptoms and physical signs preceding and accompanying the ictus are reviewed in considerable detail. There is an important chapter on investigation, in which the pitfalls associated with the examination of the cerebro-spinal fluid are discussed and the dangers and timing of angiography in cases of ruptured aneurysms are considered—the end of the first week is thought by the author to be the optimum time. Of importance is his presentation of data on prognosis: "About 20 per cent. of patients who survive an attack of subarachnoid hæmorrhage (due to aneurysm) are likely to die of recurrent bleeding, but half will do so in the first six months." After discussing the pathology of aneurysms and angiomata, he proceeds to review critically what is at present being accomplished by the neurosurgeons, concluding that with hypotensive or hypothermic anæsthesia supraclinoid aneurysms of the internal carotid artery may be most safely treated by intracranial operation, carotid ligation being reserved for infraclinoid aneurysms. Only by intracranial operation can aneurysms of the anterior, anterior communicating and middle cerebral arteries be effectively treated. The author is confident that timely surgery will prevent the fatal recurrence of bleeding which may occur within a few weeks of the ictus.

The book is attractively bound and printed. As an authoritative and comprehensive reference work it can most confidently be recommended to practitioners and physicians in search of information on subarachnoid bleeding and its management, as well as to neurosurgeons, who will appreciate the perspective a physician has brought to their field.

Cryptococcosis: Torulosis or European Blastomycosis. By M. L. Littman, M.D., Ph.D., and Lorenz E. Zimmerman, M.D.; 1956. New York and London: Grune and Stratton, Incorporated. 10¼" × 7", pp. 215, with illustrations. Price: $8.50.

INFECTIONS due to *Cryptococcus neoformans* (*Torula histolytica*) are not common. This is surprising in view of the widespread distribution of the organism on animal and vegetable material and in foodstuffs. Because no one clinician sees many of these cases, the ultimate diagnosis often comes as a surprise, especially if manifestations other than pulmonary granuloma or meningo-encephalitis have predominated; and it is quite likely that occasional cases are missed. Therefore, a book as comprehensive as this volume is especially welcome. The material includes the records of over 100 cases of cryptococcosis contributed from all parts of the world. It covers the subject from every point of view. There are excellent descriptions of infections in humans and in animals, epidemiological aspects, the pathology and the laboratory diagnosis. Little-known facets are discussed, such as the sarcoid-like lesions which are sometimes found. Yet a balanced perspective is maintained throughout.

This book can be recommended to all who are interested in fungal infections. It is well written, well illustrated and well produced. It succeeds in its aim, which is to combine the most useful features of a monograph and of an atlas in one volume.

The Principles and Practice of Medicine: A Textbook for Students and Doctors. By Sir Stanley Davidson, B.A. (Cantab.), M.D., F.R.C.P. (Ed.), F.R.C.P. (Lond.), M.D. (Oslo), and the staff of the Department of Medicine, University of Edinburgh, and Associated Clinical Units. Third Edition, 1956. Edinburgh and London: E. and S. Livingstone, Limited. 8½" × 5½", pp. 1089, with 73 figures and five colour plates. Price: 35s.

MORE strongly even than with the second edition does the impressive record of three editions and two reprints within four years argue the popularity of this book. And its very success must promote further success, as the frequency of new editions enables it to be kept fully up to date in a manner which is seldom possible in a text-book. Additionally, the cost has been kept to within half a crown of that of the previous edition, in spite of an expansion of some 38 pages and the addition of a well-deserved knighthood to the name of the principal author.

However, in spite of the expressed hope of Sir Stanley Davidson, there are variations from the uniformity which he has attempted to achieve by careful correlation of the work of the various contributors, if not so much in form of presentation as in quality, and it is to certain sections rather than to the whole that one is led to give unreserved praise. The sections on liver disease and hæmatology are particularly worthy of note. The table of normal values on the back cover will be a comfort to many. On the other hand there are a few obvious errors, notably the confusion in the section on the treatment of hypertension between methonium compounds as a class and the individual members thereof. This particular section also exemplifies one possible point of criticism—namely, that it is not always clearly indicated that the very brief account of highly specialized methods of treatment can be no more than a general outline and does not give adequate direction.

In general terms, however, the book seems certain to fulfil, even more satisfactorily than did the two earlier editions, the role for which it was designed—namely, the provision of a concise but comprehensive syllabus of lectures on the principles and practice of medicine.

Techniques in Blood Grouping. By Ivor Dunsford and C. Christopher Bowley, M.B., B.S., M.R.C.O.G., with a preface by R. R. Race, F.R.S.; 1955. Edinburgh and London: Oliver and Boyd. 9" × 5½", pp. 264. Price: 21s.

SEVERAL authoritative books have recently been published concerning the clinical use of blood transfusions. In addition, various authors have coordinated the advances which have occurred with somewhat bewildering speed in our knowledge of the human blood groups. Dunsford and Bowley have now produced the first comprehensive, but practical, manual on techniques. The pages of the manual are perforated at the binding edges and held together with two continuous wire spirals, so that the pages lie flat when the book is open on a bench. The authors claim that the bench is the correct place for the book.

The manual is divided into three sections. The first is a review of the principles on which the techniques are based The second section describes 80 techniques which are in use in the laboratories of the Regional Blood Transfusion Centre, Sheffield. The third section is a glossary of terms commonly used by blood group workers, including those who favour the Wiener nomenclature. All three sections are comprehensive and lucid, and would enable a worker without previous experience, but with a supply of materials for practice, to become proficient in blood group work, from the simplest procedures to the most specialized. Minor variations of basic techniques are separately listed for the sake of clarity, and this accounts for the large number of techniques in the second section.

The person who performs an occasional blood group test before transfusing a patient may find this book too comprehensive and detailed, and he may prefer a simpler account for his purposes. However, the hospital laboratory required to undertake a range of tests and occasionally to elucidate the unusual problem cannot afford to be without the book on the bench. The expert will find much of interest in the techniques used at Sheffield, many of which have not been previously published.

Notes on Books, Current Journals and New Appliances.

"La Guérison." Par le Dr. Jacques Sarano; 1955. Paris: Presses Universitaires De France. 7" × 4½", pp. 128.

THIS small book is a study of recovery written with a philosophical bias, primarily for non-medical readers. The whole concept of healing, recovery and cure (words with differing shades of meaning, though all represented by the French word *"guérison"*) is discussed exhaustively, in a most unusual way. The lay person is likely to find the book heavy going, though the effort is well worth while. Members of the medical profession could read it with profit and pleasure.

The Medical Journal of Australia

SATURDAY, JANUARY 12, 1957.

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THE VALUE OF USELESS LEARNING.

IN March of last year McGill University, in Montreal, Canada, conferred an honorary doctorate on the Prime Minister of Australia, the Right Honourable R. G. Menzies. Although the vagaries of Canadian weather had obliged Mr. Menzies to abandon a special aeroplane for a chilly motor-car and so brought him several hours late to the Special Convocation called in his honour, he was still able to produce a stimulating address, which had as its theme the value of useless learning.[1] The low premium often placed on what is regarded as useless learning is well known to us all. As Mr. Menzies put it: "Little Willie must as soon as possible get a licence which will enable him to charge fees to somebody." So the old questions are asked: "Why should he learn Latin? That won't do him any good. Why is he to learn ancient history? That won't do him any good. He will be just as good an operator without that knowledge." Perhaps he will, but (in Mr. Menzies's words) "he won't be anything like so good a man in the long run, because it is not a bad thing for the human mind to come across the astonishing fact that there were people in the world before us, and that, oddly enough, they had their own hopes and hates and passions and fears, as we have. And we may learn something from them". He went on to point out that we stand in the great procession of mankind, from the most remote period of history to the most remote and, of course, invisible future. "And when people understand that they are part of the procession of life then they will be all the more disposed to realize that just as what they are doing is influenced for good or ill by those who have gone before them, so what they do in their own time, in their own generation, is going to influence for good or for ill those who come after them." It is plain enough, of course, from other remarks, that Mr. Menzies fully acknowledges the value of practical studies—that is not really in question; but here his thesis is that "there is nothing like this useless learning for inducing in the mind of man a sense of continuity and of responsibility, broadening his horizons, teaching him the importance of accuracy of thinking and of generosity of heart".

This line of thought provokes a number of interesting reflections from the point of view of medical studies. It is probably sighing for the moon to hope for much greater attention to the humanities in pre-medical education. Indeed the demands of a medical course are so exacting today that one can understand the current preoccupation with the basic sciences to the sad detriment of other "useless learning" in the pre-matriculation period. At the same time one must welcome the changes being made in the matriculation requirements of the University of Sydney which are aimed at discouraging too narrow early specialization. This should help to restore the balance a little, or at least to impress on the young student the fact that the university which he is entering sets some store by useless learning.

The question then arises whether there is any place in a medical course for useless learning. The answer surely is that there is, and those who determine the curricula of our Australian medical schools are obviously aware of it. As an aside it may be remarked that medical students in Australia are at an advantage in that all our medical schools are an integral part of a university, a position which does not obtain in many medical schools overseas. The medical student has thus an excellent chance, when out of classes, of "broadening his horizons" in his pre-clinical years, even though his hospital work will take him into more sequestered paths. However, the standard medical curriculum provides at least two examples of what to many appears to be useless learning. The first is in the basic general sciences (chemistry, physics and biology) and in the pre-clinical subjects, particularly physiology and biochemistry; the second is in the history of medicine. In general the short period available in the first year for the basic scientific subjects necessarily confines the curriculum for the medical student to what he really needs as a reasonable basis for his later years; but there may be a little time for some useless learning, which will (or should) develop mental discipline and inculcate a scientific approach not immediately related to the practical issue of charging fees to somebody. A better opportunity is presented in the physiology and biochemistry departments, and here in consequence conflict of opinion becomes more apparent. Some assert that these subjects should be taught essentially as a background to clinical medicine, others that they stand entirely in their own rights and that reference to clinical medicine is only incidental. The

[1] *McGill News*, Summer, 1955.

doctrine of the value of useless learning lends considerable support to the second view, with the thought always in mind that here is an opportunity for the student to learn invaluable lessons in scientific method and the elementary requirements of research, and also to acquire a sound basic knowledge of the subjects. At the same time there is room for some compromise, as in the attitude of a discerning professor of physiology in the United States who avows firmly his intention of teaching physiology to medical students as he would to science students, as a scientific subject *per se*, but retains the thought clearly in the back of his mind that the medical student who does not make a brilliant showing may nevertheless become a good doctor. This ensures that the advantages of useless learning in the scientific field are not lost, and yet allows for tempering of the wind to the shorn lamb.

Leaving aside the opportunities for a similar approach, to which the best teachers are thoroughly alive, in such more obviously practical subjects as pathology and bacteriology, we may turn to a minor subject in the medical curriculum, the history of medicine. Medical schools value this to differing degrees, but mostly it is pushed into the background; sometimes it is quite optional, and rarely if ever is it a qualifying subject. Yet it presents a golden opportunity for the presentation of useless learning, with all the values listed by Mr. Menzies specially orientated to the medical position. The point is too obvious to be laboured, but warrants careful consideration by those to whom it is an immediate concern. However, some new and refreshing thoughts on the subject have been put forward by Lloyd G. Stevenson,[1] who, as it chances, teaches in the medical school of McGill University. He describes what happened when final-year medical students of the university were required to submit a short essay on some aspect of the history of medicine. Choice of subject was unrestricted, but purely biographical studies were known to be unwelcome. The results were interesting. Despite warning, some students undertook subjects of impossibly large scope, and others did not respond to encouragement to seek original rather than second-hand sources. On other ambitious projects Stevenson comments devastatingly: "'The yang and yin theory and its effect on Chinese medicine' was not the work of a rising young sinologist; and 'Islam, the bridge from classical Greece to the Renaissance', was not only high and unsupported in the middle but was established at either end on nothing and nought." There were, however, sound essays on a variety of subjects which gave evidence of an intelligent approach and some understanding of where to find source material. In particular Stevenson refers to the "surprising and quite exhilarating" essays that sprang from clinical interests and the "felt needs" of the students. Examples of subjects were "Changing Conceptions of Bright's Disease", "The Development of the Concept and Theory of Celiac Disease" and "Historical Aspects of Clubbing of the Fingers". Stevenson's comment is a memorable epigram: "To learn medical history in studying medicine, medicine in studying history, is a high, exciting aim." He believes that some ten or a dozen of the students achieved it, at the same time gaining closer acquaintance with the medical library. The experiment certainly seems to have been a success and worthy of emulation.

[1] *J. Hist. Med.*, 1956, Vol. II, No. 3.

All that has been said so far applies immediately to the medical undergraduate, but its implications are much wider. The medical graduate who is still interested in his continued education is invited to think them out for himself. For present purposes it is sufficient to conclude with a further quotation from Mr. Menzies's address to the Convocation at McGill University:

> A university, a great university like this, a world-famous university, doesn't exist merely to make people competent lawyers or competent engineers or competent surgeons or whatever it may be; it exists primarily to send out through its portals men and women who have touched learning by the hem and have gained something from it. A man who will never understand these things may become a mechanical genius and the enemy of mankind. What we learn here was so perfectly expressed in the opening prayer: the love of learning and the good of mankind.

Current Comment.

BEMEGRIDE, AMIPHENAZOLE AND BARBITURATE POISONING.

IN 1955 a group of Australian workers, A. Shulman, F. H. Shaw and N. M. Cass, of Melbourne, and H. M. Whyte, of Sydney, reported[1] their clinical experience in the treatment of barbiturate poisoning with $\beta\beta$-methylethylglutarimide (NP13) (later known as bemegride and marketed as "Megamide") and with 2-4-diamino-5-phenylthiazole hydrobromide or hydrochloride (D.A.P.T.) (later known as amiphenazole or "Daptazole"). The clinical trial followed on prolonged experimental study of these drugs in the laboratory. The trial covered 41 cases of pure barbiturate intoxication, of which nine were serious. One death occurred, which was considered not to be due to the treatment, and Shulman and his colleagues concluded that the method had been promising enough to warrant further large-scale clinical trials. They described the merit of the method as lying in the fact that the more active agent, bemegride, had an action unlike the central analeptics in current use; it appeared to exert, in therapeutic doses at least, a direct antagonism to the offending barbiturate, and would readily restore the patient from the deep coma to a desired state of light anæsthesia—"the safe state"—from which spontaneous recovery to full consciousness usually occurred within eight hours. That removed the need for strict and prolonged medical and nursing care and virtually eliminated the risk of complications. Amiphenazole, itself a weak barbiturate antagonist, was a good synergist to bemegride as well as an excellent respiratory stimulant. It greatly reduced the risk of toxic manifestations, which sometimes occurred when bemegride alone was given rapidly and in high dosage.

Despite certain misgivings, subsequent investigators have generally reported favourably on their experience with these drugs, most interest centring on the more powerful bemegride. Of the many single case reports published, one of the most interesting was that of A. D. F. Hurdle and B. K. Lane,[2] who treated a child, aged fourteen months. By mistake for succinylsulphathiazole, phenobarbitone had been administered to the child for thirty-six hours. On admission to hospital he was deeply comatose, but his circulation and respiration were not depressed. He was treated successfully with 2827 milligrammes of bemegride, mixed with a convenient intravenous transfusion fluid and given continuously for eighty hours. More recently A. M. Mechie[3] has reported three cases of barbiturate poisoning; two patients received bemegride and amiphenazole and responded satisfactorily, although one had ingested 34 grammes of amylobarbitone after an

[1] *Brit. M. J.*, May 21, 1955.
[2] *Lancet*, June 2, 1956.
[3] *Scottish M. J.*, September, 1956.

unknown quantity of alcohol; the third patient, who received other routine therapy, died.

The management of barbiturate poisoning is an important problem in Denmark, where, according to Carl Clemmeson,[1] the incidence of suicide has for years been high compared with that in other countries; during the period 1945 to 1951 the number of patients treated in Danish hospitals each year for barbiturate and morphine poisoning has more than doubled (from 1500 to 3000). In Copenhagen the treatment of barbiturate poisoning has been centralized in the department of psychiatry at Bispebjerg Hospital, and the resultant concentration of experience has brought about a considerable drop in mortality at the same time as the incidence of poisoning has risen. It is therefore of much interest to read four papers[2] from the Bispebjerg Hospital recording their experience with bemegride. A. Louw and L. M. Sonne have investigated its effect in the most severe cases of barbiturate poisoning among patients admitted to hospital between September and December, 1955. The series comprised 24 patients (nine male and 15 female). Most were treated with bemegride alone by intravenous infusion, apart from general and nursing treatment. Seven of the 24 patients died. Louw and Sonne state that the bemegride helped to bring about a "safe state" by stimulating reflex activity and respiration. It induced electroencephalographic changes, including patterns of universal cortical stimulation, in some cases with spikes which appeared before any clinical effect was manifest. It did not apparently shorten the period of coma, hasten elimination of barbituric acid, or cause patients to recover consciousness before the blood level of barbituric acid had fallen to that at which return to consciousness would have been expected without bemegride. Jeppe Pedersen reports the results of treating 22 patients suffering from poisoning with a barbituric acid derivative "Allypropymal", which is widely used in Denmark as a hypnotic. He states that bemegride and amiphenazole exerted an "undoubted arousing effect"; but comparison with 74 control cases of poisoning with "Allypropymal" in which these two substances were not used indicates that bemegride and amiphenazole did not curtail the period of unconsciousness or restore consciousness at higher blood levels of barbituric acid than in the controls. Carl Clemmeson makes special reference to seven patients who had respiratory paralysis with total apnœa due to barbituric acid poisoning. Each of these seven patients responded to brief treatment with bemegride and amiphenazole. Clemmeson points out that total respiratory paralysis has hitherto been regarded as an extremely serious complication of barbituric acid poisoning. Analeptics are ineffective, and as a rule death has ensued. Of 19 deaths from barbituric acid poisoning in Clemmeson's unit in 1951, five followed total respiratory paralysis. Therefore, as Clemmeson states, if only because of their effect on respiratory paralysis, bemegride and amiphenazole are valuable adjuvants in the treatment of patients with acute barbituric acid poisoning.

The last of these four papers from Copenhagen sounds a warning note right from its title: "Delirious Psychosis and Convulsions due to Megimide." In it J. Kjær-Larsen reports the occurrence of delirious psychosis dominated by visual hallucinations in 15 out of 50 patients treated with bemegride for acute barbituric acid poisoning. He states that the frequency of psychosis was greatest after administration of large doses of bemegride, and in these cases the latent period between wakening and the onset of psychosis was briefest. Nine of the cases of psychosis occurred among 12 barbiturate addicts. Among the 38 non-addicts there were six cases of psychosis; the six patients affected, who had previously been taking barbiturates in therapeutic doses, had remained unconscious for a long time after administration of bemegride. Convulsions occurred during treatment with bemegride in half the barbiturate addicts and in half the patients who developed psychoses, but in only a quarter of the non-addicted patients who did not develop psychoses. Kjær-Larsen expresses the opinion that these psychoses are "apparently exogenous reactions provoked by megimide in barbituric acid habitués. They resemble spontaneous withdrawal psychoses, but there are certain differences". He does not suggest that treatment with bemegride is contra-indicated because of the risk of psychosis, but recommends that the drug should be administered only in accordance with strict indications, particularly in drug addicts.

These observations of Kjær-Larsen are, of course, important, but it seems strange that they should appear in isolation. S. Gershon, E. M. Trautner and F. H. Shaw state, in a letter to *The Lancet*,[1] that they have reports of treatment with bemegride in over 130 cases in Europe and the United States of America, with no mention of psychoses. On the incidence rate observed by Kjær-Larsen, some 50 cases of psychosis should have been expected in the 130 cases. "Surely such observational negligence cannot be ascribed to the clinicians concerned." Attempting to explain Kjær-Larsen's observations, which they do not doubt, Gershon, Trautner and Shaw think that the cause of the psychoses should be sought in the after-effects of chronic barbiturate administration, though they are not sufficiently explained as being of a withdrawal nature. They are able to refer to a few cases in which such effects have been observed after prolonged high-barbiturate "sleep treatment". In one case these delusions and hallucinations developed in a schizophrenic patient who during an illness of four years had never shown these symptoms before. It may be noted that all Kjær-Larsen's 50 subjects were psychiatric patients. In any case, whatever the explanation of these psychotic manifestations, there seems to be no dissenting voice to the view that bemegride and amiphenazole, rightly used, have established for themselves an important place in the management of acute barbiturate poisoning.

[1] *Acta med. scandinav.*, Vol. 48, Fasc. 2, 1954.
[2] *Lancet*, November 10, 1956.

[1] *Lancet*, December 8, 1956.
[2] "Some Mechanical Factors Influencing the Distribution of Respired Gas", School of Aviation Medicine, U.S.A.F., Texas, 1956.

VENTILATION IN EMPHYSEMA.

WITHIN the past few years the work of several groups of investigators upon the mechanical aspects of breathing has brought us very much nearer a full understanding of the pathological physiology of emphysema and allied diseases of the lungs. Theoretical studies, such as that of C. B. McKerrow, A. B. Otis, R. Bartlett and B. Armstrong[2] are even proceeding more or less in harmony with experimental results. The pattern of the latter was indicated in 1951 by H. Dayman,[3] who produced good evidence for the existence of the check-valve mechanism of air-flow obstruction in emphysema. By plotting changes in intrapleural (or, for practical purposes, intraœsophageal) pressure against changes in lung volume during a respiratory cycle, a pressure-volume diagram is obtained, the area of which represents the work done upon the lungs. This work is greatly increased in emphysema, for example, where there is a considerably increased resistance to inspiratory and particularly to expiratory air flow, as has been reported by D. L. Fry, R. V. Ebert, W. W. Stead and C. C. Brown.[4] In emphysema it has been shown by J. Mead, J. Lingren and F. A. Gaensler[5] that this resistance to flow is increased by further effort, which is, of course, a most uneconomical state of affairs. They have also shown that the dynamic compliance in emphysema (compliance is the volume change produced by unit pressure change) decreases with increase in the rate of breathing—that is, the lungs become stiffer. Once again the emphysematous patient is at a disadvantage compared with the normal subject, whose compliance is unaffected by respiratory rate. Bronchodilator drugs tend to reverse the changes described, although narrowing of the bronchial lumen *per se* is not

[3] *J. Clin. Investigation*, 1951, 30: 1175.
[4] *Am. J. Med.*, 1954, 16: 80.
[5] *J. Clin. Investigation*, 1955, 34: 1005.

the only factor in their production. For the moment the reasons for these changes need not detain us, as for the most part they are of greater physical than clinical interest. They are discussed by R. M. Cherniak[1] and by McKerrow and his associates, whose ingenious theoretical analysis of ventilation (based on electrical analogies) is worthy of study by those interested in respiratory physiology. It is obvious that these difficulties must produce some limitation of breathing capacity in emphysema, but it may well be asked why mechanical factors should be stressed when in fact the prime function of the lung is to achieve gas exchange. The answer lies in the remarkable degree of dissociation between the chief pulmonary symptom, dyspnœa, and the lung's ability to take up oxygen and excrete carbon dioxide. The lack of correlation is certainly not complete, especially in "acute experiments", but remarkable degrees of oxygen desaturation and even of carbon dioxide retention are compatible with a quiet and comfortable existence. On the other hand, dyspnœa, at least in normal subjects and in patients with emphysema, asthma, pneumonia and mitral stenosis, relates fairly well to the work of breathing and perhaps more particularly to the coincident pressure changes. This view has been put forward notably by R. V. Christie[2] and his co-workers R. Marshall, R. W. Stone and B. M. McIlroy[3,4,5]; Christie has pointed out that the rate and depth of breathing normally adopted is that which is most economical in terms of work. (In passing it may be mentioned that the rate and depth of breathing are not given much stress by clinicians these days; in fact they can be very revealing in regard to the mechanical properties of the patient's lungs.) By way of comment on the relative significance of ventilation and gas exchange, it may be said that the patient would probably be more interested in knowing his intrapleural pressure swings than his diffusing capacity, despite the greater prognostic value of the latter.

Two recent papers by E. O. Attinger, M. M. Goldstein and M. S. Segal[6,7] deal with some of the problems of ventilation in emphysema and in asthma. They show a tendency to make generalizations which are not always wholly justified by their data, and they present little which is new. They do, however, stress the facts that emphysema is usually diagnosed at a comparatively late stage, that the disability is not related to radiological appearances, and that the best clue to the presence of emphysema is to be found in the study of pressure-flow relationships. It is of interest that they find a tendency for a low compliance to be associated with a low arterial partial pressure of oxygen, although in view of Cherniack's finding of a fair correlation of compliance with diffusing capacity too much significance must not be attached to this; or rather it would be better to say that a correlation does not, of course, imply a causal relationship. They advocate the more frequent performance of pressure-volume "loops" as being more sensitive, informative and reliable than the commonly performed volumetric ventilatory function tests (for example, the maximum breathing capacity, vital capacity and timed fast expiratory volumes). Their claim is a just one, and certainly it should be possible to do this in all thoracic centres. Nevertheless, the two approaches are complementary and not mutually exclusive. On the other hand, the equipment required is more than can be expected of smaller hospitals and clinics, whereas timed volume determinations are, in transatlantic terminology, "office procedures". The increased information given by the more complicated techniques must not be allowed to obscure the value of the simple tests. Their conclusion that "an evaluation of ventilatory function on the basis of volume measurements or volume-time relationships alone may be as incomplete and as misleading as evaluating cardiac function on the basis of cardiac output without taking into consideration blood pressures and vascular resistance" employs an analogy which, like many analogies, is distinctly fallacious in some respects. A good fast puff into a recording spirometer writes a most informative message to an experienced observer.

[1] *J. Clin. Investigation*, 1956, 35: 294.
[2] *Proc. Roy. Soc. Med.*, 1953, 46: 381.
[3] *Clin. Sc.*, 1954, 13: 625.
[4] *Clin. Sc.*, 1956, 15: 345.
[5] *Clin. Sc.*, 1954, 13: 404.
[6] *Am. Rev. Tuberc.*, 1956, 74: 210.
[7] *Am. Rev. Tuberc.*, 1956, 74: 220.

THE DEATH OF LOUIS XIV.

LIKE the woman in the Bible, Louis XIV in the course of his long life suffered many things at the hands of physicians; the wonder is that he did not die in early childhood. It is recorded that he had had 1500 to 2000 purgatives, several hundred enemas, pounds of Peruvian bark, and large quantities of cordials and other so-called remedies. Fagon, his chief physician, was the author of this exhausting regime; it will be recalled that he was looked upon with great disfavour by the Duchess of Orleans.[1] She never forgave him for his part in the treatment of the Duchess of Burgundy, and after the death of Louis she wrote that in her opinion, if the king had not been purged by Fagon "so often and so barbarously", he would have lived to see his eightieth year. The Duchess of Orleans was an astute observer with an acid pen. The story of the last earthly days of *le roi-soleil* is retold by R. Laulan,[2] and from the medical point of view it is of much interest. On August 10, 1715, at the age of seventy-seven years, Louis felt ill. He had an irregularly intermittent fever, and Mareschal, his chief surgeon, without making a firm diagnosis, considered the prognosis hopeless. Fagon, whose faculties had considerably declined, was blindly optimistic. On August 12 the king took his two thousand and first purgative, and later complained of pain in the left leg. Fagon said it was an attack of sciatica; but the next day a small red area appeared below his garter. The limb was rubbed with warm cloths, which procedure relieved the pain a little, and the patient was made to drink large quantities of water. Fagon gravely discussed with his colleagues the question of having the patient drink ass's milk; the decision was provisionally in the negative. Meanwhile the king, who was trying to overcome the disease process by movement, was having nights of severe pain, with a burning thirst and profuse sweating. On August 17 he took to his bed, and Mareschal, without pronouncing the word "gangrene", decided that the condition was dangerous. Fagon, finally aroused, called some of his colleagues in consultation; they all admitted that they could do nothing. They talked about sending for mineral water from Bourbon l'Archambault, but came to no decision. On August 21 the king was purged again. The next day he took a herbal bath, during which he fainted. On the same day the administration of ass's milk began. On August 23 they gave him some more, "to moisten the chest", and caused him to take another bath to give him relief, whilst the doctors discussed the virtues of ass's milk at great length; the next day it was completely out of favour. On August 24 signs of gangrene were found below the garter. They talked of erysipelas and applied compresses of camphorated brandy, without in the least deceiving the king on the nature of his illness or relieving his sufferings. Louis, giving up all hope of recovery, set about preparing for death with steadfast serenity. Nevertheless he continued patiently to submit to the experiments of his medical attendants. Once Mareschal had proved by several lancet punctures that the gangrene had reached the bone, *le roi-soleil* sent for the young Dauphin, the future Louis XV, and said a noble and moving farewell to him, whilst enduring the application of a lotion of camphorated brandy.

By this time official medicine was ready to abdicate; semi-official medicine came on the scene on August 28, represented by three quacks. The first came from Amiens, and claimed to possess an infallible remedy for gangrene. The second came from Beauvais. The third, from Marseilles, outdid the others by having himself introduced

[1] M. J. AUSTRALIA, March 3, 1956.
[2] *Presse méd.*, October 10, 1956.

by the Regent. He at once went into conference with the doctors, to whom he explained that his elixir was made from the body of an animal in much the same way as English drops were made from men's skulls. Fagon, only partly reassured, stood out against the treatment. He was roundly abused by the quack, who declared himself to be not a physician, surgeon or apothecary, but a completely disinterested person, who possessed a family secret and testimonials of cures that he had accomplished. Mareschal, consulted by his colleagues, sensibly announced that he had no faith in the proposed cure, but that there was nothing to lose by trying everything. The quack was taken to the bedside of the king, who was resigned to dying and unwilling to try any new experiment; he felt the patient's pulse, announced that there was still hope, and prudently indicated that he could not guarantee a complete cure, as it was rather too late. He poured a few drops of his "elixir of life" into a small glass of burgundy. The king, disgusted by the odour, at first refused the potion, but finally drank it, saying: "I take it neither with hope nor with the desire of being cured; but I know that in the state in which I now am, I must obey the doctors." (How exemplary a patient was this!) A strange sequel was that after a second dose the patient was calmer and able to see more clearly and to speak more strongly. Further doses of the elixir had the same effect—that of a few drops of oil in a lamp that is going out. The king, who was hovering between life and death, regarded himself as already out of this world, and spoke of his successor as "the young king". At 8 o'clock on August 29 he swallowed the last doses. The gangrene had reached his knee, and even his thigh was swollen. On August 30 the limb was as foul as that of a corpse, and at 5 o'clock on the morning of September 1 *le roi-soleil* piously yielded up his spirit after the successive onslaughts of the physician, the surgeon and the quack.

This is a dramatic story of royal fortitude and medical ineptitude; all that can be said of the doctors is that they certainly did their best. Laulan remarks that during the twenty-three days of his final illness, Louis XIV gave one of the noblest performances that a man could give. "He entered into eternity with that majestic and easy step with which he was wont to cross the Hall of Mirrors before all the bowed heads."

ORAL POTASSIUM THERAPY AND SOME SOURCES OF POTASSIUM.

In recent years the importance of the potassium ion in physiological processes has become more and more reflected in clinical practice. Removal of potassium is sometimes necessary, as in some cases of prolonged anuria. Primary alderosteronism (not to mention the commoner secondary condition) and potassium-losing nephritis are relatively rare conditions in which potassium supplements are required; steatorrhœa and hepatic cirrhosis may also be associated with potassium depletion. In practice, perhaps the commonest situation in which potassium deficiency is likely to arise is during long-continued treatment with mercurial diuretics of patients with congestive cardiac failure. As a prophylactic measure, many physicians replace one gramme of the three grammes of ammonium chloride commonly given with one gramme of potassium chloride. As anyone who has taken either substance knows, this treatment is unpleasant. C. T. G. Flear, Molly Hill and G. Thomas[1] list the symptoms with which oral potassium therapy may be associated: epigastric discomfort and burning, nausea, vomiting, meteorism, diarrhœa, stiffness of the fingers and a metallic taste in the mouth. These sequelæ are commoner in the more seriously ill patients, and they appear to be independent of dosage and of serum potassium level. Having made these observations, and been troubled by the patients who repeatedly vomited their medicine, they tackled the problem in two simple, practical ways. They first tried eight different preparations of potassium given to 32 consecutive patients with congestive heart failure, commencing with potassium chloride. Five patients had no symptoms with this, and 13 tolerated it despite some symptoms. For 11 of the remaining 14 patients some tolerable preparation of potassium was found, although seven had persistent symptoms. The most satisfactory form of administration was "potassium effervescens". This was a tablet composed of potassium bicarbonate 26·56 grammes, tartaric acid 16·87 grammes, citric acid 11·25 grammes and sucrose 9·4 grammes per 100 tablets, the mixture being granulated on a hot-plate. Five of these contain 13·3 milliequivalents of potassium. The other substances tried were potassium chloride with calcium carbonate, potassium bicarbonate alone, potassium dihydrogen phosphate, di-potassium hydrogen phosphate (the last two will not keep in solution), potassium citrate and Imperial water.

The second practical approach to the problem was to analyse natural and commercially available fluids for their potassium, sodium and chloride content. In making their survey they have cast their net widely, for a variety of beers, ciders, wines, fruit juices and squashes, and some other items are listed. In fruit juices such as orange, grapefruit and pineapple, there are about 40 milliequivalents of potassium per litre, with a sodium content of less than one milliequivalent; the fruit squashes and soft drinks are useless. Patients who like their Guinness will have their faith vindicated to some extent, for it contains 15 milliequivalents of potassium and 1·5 milliequivalents of sodium, rendering it the most satisfactory of the lighter alcoholic beverages in this respect. Unfortunately, the quantities stated are per litre. Some of the red wines have a more favourable proportion of potassium to sodium and double the potassium content of Guinness, but a litre is possibly more than one would wish to prescribe, even if a benevolent government provided it free. It is, however, plain that dietetic sources could provide part of the potassium intake, with benefit to the patient's comfort.

In these columns it has frequently been noted that there are many useful research projects in medicine which do not need elaborate equipment or esoteric statistical analyses to be of considerable value. This is one such investigation. It has resulted from the collaboration of the medical registrar, the house physician and the biochemist in a Birmingham hospital, all of whom have been prepared to give some time and thought to the practical solution of a practical problem. If we but paused to think, we would find similar problems every day.

[1] *Lancet*, December 8, 1956.

AN AID IN THE CHANGING OF TRACHEOTOMY TUBES.

A USEFUL SUGGESTION is offered by Gabriel Tucker[1] to those who have difficulty in the changing of tracheotomy tubes. A length of tubing, such as a segment of urethral catheter, of uniform outside diameter and an inch or two larger than twice the length of the tracheotomy tube, is selected as a guide to the tracheal opening. Its outside diameter is approximately the same as that of the inner tracheotomy tube, so that when lubricated it should pass readily, but not loosely, through the outer cannula of the replacement. The tube is cut with a short bevel on one end and a long bevel on the other. To change the tracheotomy tube the inner tube is first removed, and the rubber guide is introduced in its place. The outer tracheotomy tube is then removed with one hand while the other hand keeps the guide tube in place. The replacement tube without its inner cannula is threaded over the long bevel of the rubber guide tube and advanced into position. The guide tube is withdrawn, and the inner cannula of the replacement tube is inserted. The method is described as being specially useful where the fistula is abnormally long or irregular, or for small children or for a patient in a respirator, where space to manœuvre is limited.

[1] *Arch. Otolaryng.*, July, 1956.

Abstracts from Medical Literature.

OPHTHALMOLOGY.

Retinal Detachment.

J. W. OKAMURA AND C. L. SCHEPENS (*Sight Saving Rev.*, Volume XXXV, 1955) describe the operation performed most frequently in treating retinal detachment at the Retina Service of the Massachusetts Eye and Ear Infirmary. This operation is a scleral buckling operation. Pre-operatively bed rest is not prescribed, and post-operatively early ambulation has not proved harmful. The operation is performed under general anæsthesia. After localization of tears a lamellar scleral resection of an area, five millimetres in width and usually involving one-half the circumference of the globe, is performed. Surface diathermy is then lightly applied to the thinned sclera, and black silk mattress sutures are placed across the thinned-out section; after this fluid is evacuated by one or more perforations through the thinned area. A piece of polyethylene tubing is inserted under the sutures, which are then tied. Diathermy is then applied in a double row from each end of the buckle to the region of the *ora serrata*. Muscles are then reattached, and the conjunctiva is sutured.

Peripheral Iridectomy.

O. BARKAN (*Am. J. Ophth.*, June, 1956) describes a new technique for peripheral iridectomy with the purpose of retaining the anterior chamber at the time of operation as well as preventing later leakage from the wound. The objective is accomplished by making the incision vertical to the sclera 1·25 millimetres posterior to the corneo-scleral border in the 10.30 or 1.30 meridian and no longer than one millimetre where it enters the anterior chamber. This small incision is plugged with iris before aqueous can escape, and the chamber is retained. The author describes the details of his technique.

Ocular Sequelæ of Tuberculous Meningitis.

A. J. MOONEY (*Am. J. Ophth.*, May, 1956) reports on the eye sequelæ met with in 65 cases of tuberculous meningitis. Of these cases 47 were associated with ocular lesions. Choroidal tubercles were present in one-quarter of the cases, and their presence can be taken as evidence of miliary tuberculosis. New tubercles appear when the disease is active, and with disappearance of the pyrexia no fresh tubercles appear. If they are increasing in number, both neurosurgery and the use of cortisone or ACTH are contraindicated. Papilloedema was present in 17 of the 65 cases. The author has the impression that there are two types of papilloedema: one an œdema of the nerve head not necessarily associated with raised intracranial pressure, and the other a typical papilloedema. He reports four cases in which there was gross papilloedema in which surgery was performed to clear the optic nerves and chiasma of exudate and so relieve the internal hydrocephalus and save vision. Pallor of the disks with clear margins was present in 13 cases. The author reminds the reader that it is difficult to assess the visual acuity from the appearance of the disk. Eight patients were operated upon with the view to removing adhesions which, it was thought, might be interfering with the blood supply to the chiasma. The author considers that, provided the patients' general condition is satisfactory, then surgery is warranted if it is thought that vision may be saved. If cortisone is to be of value it should be given very early in the course of the disease and continued for three weeks.

Atrophy of the Stroma of the Iris.

P. A. CHANDLER (*Am. J. Ophth.*, April, 1956) discusses essential progressive atrophy of the iris and reports on six cases which were somewhat different from the classical form of the disease. The syndrome described by the author is characterized by slight atrophy of the iris stroma, peripheral anterior synechiæ, mild glaucoma, dystrophy of the posterior corneal surface and corneal œdema. In the cases reported the atrophy of the iris was slight and confined to the stroma, the pupil had remained central and round, glaucoma although mild had appeared early, and there was marked corneal dystrophy with corneal œdema. The disease was unilateral in all cases. The author recommends an early filtering operation to relieve the glaucoma and corneal œdema.

Prednisone and Prednisolone in Ocular Disease.

D. M. GORDON (*Am. J. Ophth.*, April, 1956) reports on the use of prednisone and prednisolone and compares these drugs with other steroids such as cortisone and hydrocortisone. For local therapy a 0·25% suspension of either drug was considered optional, while for ointment 0·5% strength was considered best. In the cases treated mydriatics and antibiotics were not used except when synechiæ were present. Eleven out of 16 patients with anterior uveitis gave an excellent response within two days after the beginning of treatment and were symptom-free within five to seven days. One drop of the suspension was instilled into the eye every two hours, and the ointment was instilled on retiring. In three of the five failures the patients showed an excellent response to systemic therapy with prednisone or prednisolone. In all cases of conjunctivitis treated improvement occurred without antibiotics. In general prednisone and prednisolone appeared to be more effective than cortisone or hydrocortisone in the management of the same conditions. For systemic therapy 40 milligrammes were given daily in severe cases, and in less severe cases 30 milligrammes were given. The indications and contraindications for prednisone and prednisolone are the same as for the other steroids.

Streptokinase and Streptodornase for Hypopyon Corneal Ulcers.

L. AGARWAL AND S. R. MALIK (*Am. J. Ophth.*, May, 1956) present the results of treatment of severe hypopyon corneal ulcer with streptokinase. Every eye treated with streptokinase was saved. In addition atropine and heat were applied, and erythromycin was given both locally and orally. Streptokinase was used as a drop in strength of 4000 units per millilitre with 1000 units of streptodornase instilled every one or two hours.

Retrolental Fibroplasia and Oxygen Concentration.

D. H. WEINTRAUB *et alii* (*J. Pediat.*, July, 1956) have studied the effect of oxygen on the incidence of retrolental fibroplasia. Premature babies were divided into two groups; one group was maintained in an oxygen concentration of 60% to 64% for thirty days, while the other group remained in 30% oxygen for thirty days. The infants varied in weight from 1075 to 2000 grammes. A total of 83 infants was observed. Eye examination was commenced between the second and third weeks after birth and continued at weekly intervals until the babies were discharged from hospital, when the weight was 2500 grammes. After discharge from hospital infants were examined at monthly intervals. Examination was performed with the pupil dilated and the infant wrapped in a blanket. After six months anæsthesia was occasionally necessary. Of the total number kept in the high oxygen concentration, 45% developed retinal vascular changes, 36% partial cicatricial changes, and 16% complete cicatricial changes or blindness; of the infants kept in the low oxygen concentration, 9% developed retinal vascular changes, 3% partial cicatricial changes, and none complete cicatricial changes. Both eyes were involved in an identical process in the affected infants. Since this study was carried out premature infants who are cyanotic from respiratory distress have been given oxygen up to 38% for periods varying from twelve hours to seven days; they are then transferred to room air. With the use of this routine there has been no case of blindness. The restriction of oxygen therapy to definite needs has exerted no deleterious effect on mortality rate. The authors also note that with restricted use of oxygen the incidence of hyaline membrane disease in premature infants has lessened.

OTO-RHINO-LARYNGOLOGY.

Ménière's Disease.

H. M. GOODYEAR (*Arch. Otolaryng.*, April, 1956) suggests that perhaps 50% of all cases considered as Ménière's disease prove, on further careful examination, to be of cardio-vascular origin. A preliminary classification can usually be arrived at through a simple history. Dizziness which occurs together with a feeling of blurring of vision is usually of circulatory origin. An increase of symptoms on stooping may indicate a very high or very low blood pressure, or some general illness. Dizziness which ceases on closing the eyes suggests an ocular disturbance. A sensation of giddiness without nausea or ataxia may be attributable to cerebral otosclerosis and is most frequent in patients past sixty years of age. Dizziness may occur with

infection of the sphenoid, and this may be relieved by cocainization of the sphenopalatine ganglion and shrinkage of the tissues at the *sinus ostium*. Dizziness which produces a sensation of objects moving about the patient indicates a labyrinthine origin. This may occur particularly when the patient first lies down, turns in bed, or rises in the morning. If attacks are sudden and severe, with pallor, sweating, nausea and vomiting, accompanied by the feeling of deafness and tinnitus in one ear, Ménière's syndrome is reasonably certain. Recurrent attacks are diagnostically important. A caloric test will reproduce the sensations experienced. Nystagmus is towards the affected ear. Recruitment can usually be demonstrated. In acoustic tumours there is no recruitment, and there is likely to be a diminished corneal reflex on the affected side. In the relief of symptoms focal sepsis must be considered. Extraction of diseased teeth and cure of a diseased sinus, gall-bladder or prostate are important. Alcohol or tobacco may be the cause. Inflation of an obstructed Eustachian tube may give relief. The control of any allergic tendencies with antihistamines, and release from worry and tension may help. A severe Ménière's attack can be relieved within ten to fifteen minutes when atropine sulphate (1/150 to 1/75 grain) is given by the hypodermic route. Nicotinic acid (50 milligrammes) *plus* "Bellergal" after meals, ergotamine tartrate and phenobarbital at bed time, may be helpful. Histamine given intravenously is worth a trial. Should a patient have frequent attacks of Ménière's syndrome with tinnitus and considerable loss of hearing in one ear, increasing with each attack, then after a fair trial with medicinal measures surgical treatment is indicated. Through a mastoid burr hole the external semicircular canal is opened, and its membranous contents, together with the ampulla and adjacent utricle, are destroyed with a fine dental explorer.

Transtympanic Mobilization of the Stapes.

C. M. KOS (*Ann. Otol., Rhin. & Laryng.*, December, 1955) states that when a loss in air conduction acuity for pure tones reaches 50 to 60 decibels, the stapes is generally considered to be firmly fixed, at least in so far as sound pressure excitation is concerned. When the air-bone conduction thresholds do not differ by more than 30 decibels, the stapes may be partially mobile. Stapes mobilization may be useful in patients with incomplete ankylosis and in those in whom the bone conduction thresholds are so elevated as to preclude a successful fenestration operation. Access is gained to the tympanic cavity via the external auditory canal. A flap of integument is reflected off the postero-superior canal wall in the direction of the tympanic annulus. The separation of the flap is then carried inwards, the annular ligament and tympanic membrane being lifted from the annular groove. The long process of the incus and incudo-stapedial articulation are thus exposed to view. With a stapes mobilizer, pressure is applied backwards to the head and neck of the stapes, in an attempt to cause the ankylosed stapes to be released so that it may function again. The reflected margin of the tympanic membrane and membranous canal wall are replaced, and are firmly healed in position in five to seven days. The technique is effective. No clear criteria have become evident for the selection of cases. Analysis of 65 cases seems to confirm an observation that the best results are obtained in the early stages of ankylosis. Regression of the hearing improvement occurs in patients with incomplete ankylosis, as well as in those with complete ankylosis, but more frequently in the former. Regression may occur within one to six months of operation. The operation may have its most effective application in that group of cases in which bone conduction thresholds are elevated to such an extent as to preclude a successful fenestration operation. In these ears, the hearing may be restored to the level of the bone conduction threshold. The technique employed does not compromise the success of subsequent fenestration. Complications of the operation are negligible. Results in general have not been sufficiently encouraging to justify complete acceptance of this operation. Criteria for selection are needed. A little fewer than one-third of the 65 ears operated on gained in hearing to levels of 30 decibels or better. No improvement occurred in 31 cases. The hearing was made worse in only one patient. Regression occurred in four.

Atresia of the External Auditory Canal.

M. W. SIMMONS (*Arch. Otolaryng.*, February, 1956) suggests that atresia of the external canal may be congenital or acquired. Acquired atresia may be caused by soft-tissue or bony disease, or by injury. Congenital atresia presents the more complex problem. If it is bilateral, a severe hearing impairment exists from birth with consequent tardy development of the child. A speech defect is coexistent. Congenital atresia of the canal without deformity of the pinna is seldom seen. Unilateral atresia is more common. With atresia of the external auditory canal the eardrum is probably rarely normal. The tympanic membrane may be in the form of an occluding plate of bone. Frequently scar-tissue bands are present within the tympanic cavity. The malleus and incus may be fused. A conduction type of deafness is invariably present with obstruction of the ear canal by atresia, either congenital or acquired. It is important to determine any associated cochlear type of hearing loss. Early surgical correction of bilateral congenital atresia with normal cochlear function is advisable. In unilateral cases, correction may be desirable for cosmetic reasons. A 20 to 30 decibel loss of hearing will remain even after a satisfactory operative result. Endaural exposure is best, the incision being always anterior to the pinna; a watch is kept for a likely malposition of the facial nerve. With the electric bone drill the cortex of the mastoid is removed and the antrum is entered. The lateral semi-circular canal can then be identified. An apparently normal eardrum should be interfered with as little as possible, to avoid scarring. Fused ossicles may need to be removed. If the stapes is fixed, a fenestration may be possible at a second stage. A hollow polythene tube of appropriate size for the new external auditory canal is selected. A split-thickness skin graft is wrapped around the lubricated tube, which is then fitted snugly into the newly made bony canal. The hollow tube affords excellent drainage and may be allowed to remain for three or four months, or indefinitely if required. Cosmetic correction of the deformed auricle may be undertaken surgically, or a prosthesis may be supplied.

MEDICINE.

Cineradiography and Electrocardiography.

M. PRINZMETAL, R. KENNAMER, S. M. WEINER AND J. R. BISHOP (*J.A.M.A.*, July 28, 1956) describe a technique of simultaneous cardiac cineradiography and electrocardiography. A fluoroscopic screen is used, the image being intensified by means of a special piece of apparatus between the patient and the screen. The electrocardiographic image is conveyed by means of an oscillometer, a cathode ray tube and a system of lenses and mirrors, to a point between the camera and the fluoroscopic screen. When the photograph is taken, the electrocardiographic tracing and the image on the screen are superimposed. The cinematic picture is taken at a rate of up to 55 frames per second. A film of 35 millimetres is used. This is reduced to 16 millimetres for projection. It has been found that a better image is obtained by this means. During the photography and electrocardiography, the electrode is placed on the chest wall as near as practicable to the part of the heart that is being studied. When the picture is projected the *tempo* may be slowed down, so that fourteen seconds may be used to study action that took only one second to photograph. The authors discuss various uses to which the method may be put in the study of cardiac disease. They consider that, as technique and apparatus are improved, more information will be obtained.

Reducing Diets.

A. KEKWICK AND G. L. S. PAVAN (*Lancet*, July 28, 1956), in a study of weight-reducing diets, found that when the Calorie-intake was constant at 1000 per day, the rate of the patient's weight loss varied greatly according to the composition of the diet. It was most rapid with diets of high fat content and less rapid with diets of high protein content, while weight could be maintained for short periods if the diet was chiefly in the form of carbohydrates. At a level of intake of 2000 Calories a day, weight was maintained or increased in four out of five obese patients, but weight loss occurred in these same subjects when Calorie intake was raised to 2600 per day, provided that this was mainly in the form of fat and protein. The weight lost on these diets appeared to be partly derived from the total body water (30% to 50%) and the remainder from the body fat (50% to 70%).

British Medical Association News.

SCIENTIFIC.

A MEETING of the New South Wales Branch of the British Medical Association was held on September 13, 1956, at Sydney Hospital, Sydney. The meeting took the form of a number of clinical demonstrations by the members of the honorary medical staff of the hospital.

Retrobulbar Tumour as a Cause of Atypical Facial Pain.

DR. J. W. LANCE showed a married woman, a pensioner, aged sixty-eight years, who had been referred by Dr. C. Cunningham, of Hurstville. For four years the patient had suffered from severe facial pain, which occurred in attacks, usually at about 12 midnight, and lasted for about two hours. It occurred less commonly in daytime, about every second or third day. The site of the pain was at first in the right naso-labial fold, then it radiated to the right eye, then over the entire right side of the head. She was nauseated with the pain, but did not vomit. There was no flushing of the face, but she noticed that her eye became more prominent with the attack. On occasions it even protruded far enough to touch her glasses. Aspirin, phenacetin and caffeine relieved the pain to a minor degree only. On being questioned, the patient said that her vision was poor in the right eye and had been so for about twelve years. She said that her sight had failed suddenly in that eye over a matter of minutes twelve years previously and had never fully recovered.

On examination of the patient, her visual acuity was 6/9 in the left eye (corrected), but only 3/60 (corrected) in the right eye. The optic disk was pale on the right side, and the visual field was constricted in the right eye. She had mild bilateral nerve deafness. The right eye was noted to be prominent, and when the projection of both eyes was observed from above the patient's head, the right eye could be seen to protrude a few millimetres more than the left. Slight resistance was felt when the right eye was pushed back into the orbit. There was no intracranial or orbital bruit, and no other abnormality was found in the nervous system. Examination of other systems revealed no other abnormality apart from a blood pressure of 220 millimetres of mercury, systolic, and 110 millimetres, diastolic, and a thrusting left ventricular type of apex beat. She was considered to have a retrobulbar mass, either a benign tumour or mucocele, causing her attacks of atypical facial pain.

The patient was admitted to Sydney Hospital, and plain X-ray films of the orbit and optic foramina showed no abnormality. Orbital exploration was performed by Dr. G. Arthurs, and a rounded tumour approximately two centimetres in diameter was found. This was successfully removed, and histological examination showed it to be a neurofibroma. Dr. Lance said that the patient had recovered well from the operation, but had a residual sixth nerve palsy. She had had no further attack of pain; her visual acuity was improving in the right eye, and was 6/36 at the time of the meeting, although less than one month had elapsed from the time of operation. Dr. Arthurs considered that the tumour arose from the sixth nerve, and it was very doubtful whether the patient would recover abduction movement in the right eye. However, she felt that that was a small price to pay for the relief of her intense and disabling facial pain.

Cavernous Sinus Syndrome.

DR. L. S. BASSER showed a woman, aged seventy-one years, who some six months previously had noticed the gradual onset of drooping of the left upper eyelid; the condition progressed until the lid had dropped completely in a matter of a few weeks. That was accompanied by some aching pain in the left eye. She noticed when she raised the left lid with her hand that she saw double, but was not as a rule troubled by it, as the lid completely covered the eyeball. Some two months later she noticed occasional paræsthesiæ over the forehead and left cheek, which became numb.

On examination of the patient when she presented at the neurology clinic, she was a pleasant, cooperative woman who gave a clear account of her disabilities. There was complete ptosis of the left upper lid, and complete external ophthalmoplegia, the eye resting in the mid-position in regard to both horizontal and vertical axes. The left pupil was dilated and larger than the right, and did not react to light, direct or consensual, or on attempted accommodation convergence. There was, however, a consensual light reflex in the right eye when the left retina was illuminated. The left corneal reflex was absent, and she did not feel the stimulus of cotton-wool on the cornea. There was gross impairment of all sensory modalities over the distribution of the first and second divisions of the trigeminal nerve on the left. No bruit was audible. The visual fields were full and the fundi were normal. No other abnormality was found on neurological examination. Her blood pressure was 200 millimetres of mercury, systolic, and 110 millimetres, diastolic, and general examination revealed no other abnormality. A provisional diagnosis was made of a lesion within the left cavernous sinus involving all three oculomotor nerves, III, IV and VI, and the first and second divisions of the trigeminal nerve. The history of onset suggested a slowly progressive lesion, and the possibility of an intracavernous carotid aneurysm was considered. A plain X-ray film of the skull revealed a crescent of calcification in the region of the left cavernous sinus, which strongly supported the diagnosis. The early involvement of the third nerve and the internal ophthalmoplegia were of interest.

Dr. Basser said that subsequently there had been some improvement. Sensation had returned over the first and second divisions of the trigeminal nerve, and no impairment could be detected on clinical testing, the only residuum being an absent left corneal reflex. Ptosis was still complete. The eye at rest was now slightly abducted, and a small range of movement was possible in all directions. The pupillary changes remained unaltered. The patient had remained well, and there had been no other change in the physical signs. The course of her condition was in accord with an infra-clinoid aneurysm, and the involvement of the first and second divisions of the trigeminal nerve suggested that it was chiefly in the middle of the sinus (Jefferson). With regard to the prognosis, Dr. Basser discussed the possibility of a carotico-cavernous fistula forming, and mentioned the rarity of subarachnoid hæmorrhage from such aneurysms, owing to the protection of the dural walls of the sinus. In differential diagnosis, he referred to carcinoma of the naso-pharynx, which might produce a similar syndrome, and to the possibility of a lateral extension of a pituitary tumour into the cavernous sinus. There was no evidence of either of those conditions in the case under discussion.

Recent Therapeutic Measures in Hypertension.

The members of the cardio-vascular clinic, DR. F. RITCHIE, DR. G. E. BAUER, DR. M. H. WHITE and DR. G. MICHELL, discussed some of the newer ganglion-blocking agents in the treatment of severe and malignant hypertension. It was stated that the clear-cut indications for those drugs were the following: (i) accelerated (malignant) hypertension with severe retinopathy, grade III or IV; (ii) some hypertensive symptoms, left ventricular failure, paroxysmal nocturnal dyspnœa, recurrent pulmonary œdema; (iii) a rising diastolic pressure exceeding 130 millimetres of mercury in a young subject, especially male. Relative contraindications were: (i) renal failure with a blood urea level over 100 milligrammes per 100 millilitres on repeated examination; (ii) cardiac infarction and major cerebral vascular accident (for six weeks); (iii) an age over sixty years, especially if there was evidence of cerebral vascular degeneration. The ganglion-blocking agents at present available included the following: hexamethonium ("Vegolysen"), pentolinium ("Ansolysen"), "Ecolid" (Ciba), "Presidal" (Burroughs Wellcome), mecamylamine ("Inversine"). It was pointed out that all those compounds had similar pharmacological actions, but differed somewhat in their side-effects. Some were also better absorbed from the gastro-intestinal tract than others. The members of the clinic then described their experience with one of the new ganglion-blocking agents, "Ecolid" (Ciba). Eighteen patients had been treated, 12 receiving the drug orally, six by injection. "Ecolid" was found a potent and useful hypotensive agent, much more reliable when given parenterally. Other new drugs were at present being tried.

Anæmia due to Diaphragmatic Herniation.

DR. R. L. WALKER showed a married woman, aged seventy-five years, who had been under the care of the hæmatology clinic for the past eight weeks, her main symptoms being dyspnœa on effort, dizziness and dyspepsia (nausea, occasional vomiting, epigastric discomfort, flatulence). Symptoms had been present for about one year, and when they first developed, the patient, then a traveller in England, had been treated at St. George's Hospital, London. A hæmoglobin level of 30% had been found, and a blood transfusion had been given. The hæmoglobin level had increased to

70%, but some six weeks later it had fallen to 45%. A large diaphragmatic hernia had been found on X-ray examination, but no treatment had been given. On her return to Australia the patient presented herself at Sydney Hospital for treatment, and was referred to the hæmatology clinic, where the following results were obtained: the hæmoglobin value was 5·9 grammes *per centum*, the anæmia being hypochromic and microcytic in type; occult blood was present in the fæces; and a large indirect diaphragmatic hernia was found on X-ray examination, which was present even when the patient was in the erect position.

Treatment consisting of "Amphojel", "Nulacin" milk tablets, and daily intramuscular iron injections ("Imferon") was given, and a steady improvement had occurred over the eight weeks of treatment. Occult blood disappeared from the fæces after four weeks, the hæmoglobin level increased from 5·9 to 13·3 grammes *per centum*, and symptoms disappeared. After four weeks the intramuscular iron injections were reduced to three times weekly, and then suspended in favour of orally administered iron (ferrous gluconate). Dr. Walker said that surgical treatment had been rejected because of the patient's age.

Sarcoidosis.

DR. E. H. HELLER showed a married woman, aged thirty-eight years, who had presented with the complaint of swelling and stiffness of the ankles and feet of about nine months' duration, with exacerbation of the pain during the night. She also complained of exertional dyspnœa, cough, and mucopurulent sputum, which she expectorated mainly on waking in the morning. She had had bronchiectasis for many years, diagnosed after a bronchographic examination. A "leaking valve" was said to be present.

On examination of the patient, her pulse rate was 80 per minute, her blood pressure was 160 millimetres of mercury, systolic, and 110 millimetres, diastolic, and the heart was not clinically enlarged. A faint systolic apical murmur was audible. Examination of the respiratory system revealed widespread râles and rhonchi, more pronounced at the bases of the lungs. There was no evidence of diminished air entry. The liver was enlarged three fingers' breadth below the costal margin, and was smooth and tender. Examination revealed pitting œdema of the lower extremities. There was periarticular swelling of the wrists only. A number of investigations were carried out. X-ray examination of the chest revealed an increase in the markings at the bases of both lungs; the appearances were consistent with underlying bronchiectasis. X-ray examination of the extremities revealed no bone or joint lesion around the knees, wrists or feet. Pathological examination of the sputum revealed numerous pus cells, but no organisms and no acid-fast bacilli; culture produced a growth of *Neisseria catarrhalis*. The hæmoglobin value of the blood was 8·5 grammes *per centum*; normocytic hypochromic anæmia was present.

The patient was treated by the administration of expectorant cough mixtures, penicillin, "Butazolidin", aminophyllin and "Polyhæmin", and with physiotherapy; her symptoms decreased. At the time of the meeting she was well, except for mild exertional dyspnœa and severe joint deformities. Dr. Heller said that a recent repetition of the investigations had yielded the following additional information. X-ray examination of the chest had revealed changes compatible with bronchiectasis in the right cardio-phrenic angle. Slight cardiac enlargement was evident; it appeared to be mainly in the right auricle and ventricle, but was minimal in degree. A bronchographic examination showed that the bronchiectasis affected the anterior basal and lateral bronchi on the right side and the interior lingular bronchus on the left side. In the extremities there were pronounced gouty changes affecting the bones of the hands, wrists and feet. The serum uric acid content was 3·1 milligrammes, the serum calcium content 9·3 milligrammes, the blood urea nitrogen content 8·0 milligrammes, and the blood creatinine content 1·0 milligramme, all per 100 millilitres; the urea standard clearance was 145%, and the serum alkaline phosphatase content was 10·3 King-Armstrong units. Liver function tests showed that the thymol turbidity was less than two units and the zinc sulphate turbidity was four units. The total serum protein content was 6·8 grammes *per centum* (albumin 4·4 grammes, globulin 2·4 grammes). An electrophoretogram gave the following results: α_1 globulin 42·7%, α_2 globulin 15·7%, β globulin 18·9%, γ globulin 23%. A blood count gave the following information: the hæmoglobin value was 9·0 grammes *per centum*, the hæmatocrit was 32% and the mean corpuscular hæmoglobin concentration was 28%; the blood picture was that of microcytic hypochromic anæmia. The Mantoux test produced a positive result.

Dr. Heller discussed the criteria in favour of a diagnosis of sarcoidosis. He said that the plain X-ray film of the chest tended to show a reticular increase in lung markings, although the appearances in the right cardio-phrenic angle were in keeping with the diagnosis of bronchiectasis. In the X-ray films of the hands and feet two types of lesions were seen: (i) multiple punched-out areas surrounded by relatively normal bone; (ii) widening of the bone with thinning of the cortex and osteoporosis. The changes were apparently typical of the radiographic changes seen in sarcoidosis. The hepatomegaly might be due to sarcoidosis, but it might also be partly due to congestion. Another diagnostic feature was the increase in the serum globulins, which affected mainly the α_2 and β fractions, but also the γ fraction. The positive response to the tuberculin test was not necessarily against the diagnosis of sarcoidosis, although some authorities held that unless the result of the test was negative, the diagnosis of sarcoidosis was untenable. Further tests necessary to confirm the diagnosis were liver biopsy, or scalene node biopsy, or biopsy from one of the bony lesions. Other diagnostic possibilities included rheumatoid arthritis with bronchiectasis and congestive cardiac failure.

Calcinosis.

DR. R. F. ROSSLEIGH showed a single woman, aged fifty-six years, who had first attended the medical out-patient department in September, 1948, complaining of intermittent pain in the left shoulder, of twelve months' duration. She had a history of diphtheria at the age of seven years and jaundice (possibly infective hepatitis) at the age of nine years. In 1938 she had undergone partial hysterectomy and right oophorectomy. The family history was irrelevant. Her sister said that the patient had always been somewhat mentally retarded, and that her pronounced loss of hearing dated from the time when she had diphtheria.

Examination of the patient revealed no abnormality in the cardio-vascular system, the central nervous system or the respiratory system. Radiological examination revealed "definite arthritic changes in the articular facets of the shoulder joint"; no other abnormality was seen. The patient was referred to the orthopædic department for treatment. She reported next in September, 1952, complaining of numbness and cold feelings in the hands and feet. Her fingers would go white and stiff for many minutes, and she felt "pins and needles" in them. A diagnosis of Raynaud's syndrome was made, and her hands were radiologically examined; the radiologist reported the presence of shadows in the soft tissues, due to calcinosis.

In March, 1953, she was admitted to hospital with hæmatemesis and melæna, the source of which was never found. It was thought that she probably had an acute gastric ulcer due to her great consumption of salicylates. From March, 1955, hard nodules, which she had noticed some time previously round the left elbow joint, began to ulcerate, and became secondarily infected. Several X-ray examinations at that stage showed the wide extent of the calcinosis.

Porphyria.

DR. J. H. E. VOSS showed a male patient, aged forty-five years, whose presenting symptoms were an "anxiety state", loss of weight and recurrent pain in the limbs and abdomen. He presented in a state of acute anxiety over the possibility of developing tetanus, because he had scratched his head with a dirty finger nail. For the previous six months a variety of symptoms had prevented him from following his trade, that of a printer. He had experienced "queer feelings" and recurring stabbing pains in his arms and shoulders, in the hypogastrium and in both loins. During the previous six months he had lost over one stone in weight. During his wanderings from doctor to doctor for relief of his symptoms, the possibility of lead poisoning was excluded. A psychiatrist treated him with a course of hormone injections, which made him worse. Phenobarbital, which was prescribed frequently, made him feel "peculiar". After he had taken a "Veganin" tablet, his wife noticed that he behaved in a strange manner. On several occasions he had noticed that the urine he passed was very dark in colour.

On examination, the patient was a thin, anxious-looking man with a peculiar detached manner, which bordered on the psychotic. There was a slight tremor of the hands, which were dry. The thyroid gland, pulse rate and eyes were normal. His blood pressure was 100 millimetres of mercury, systolic, and 60, diastolic. The remainder of the physical examination revealed no abnormality. The specimen of urine presented was a deep port-wine colour, and on investigation the urine was found to contain porphyrins and porphobilinogen.

Dr. Voss said that, despite the withholding of drugs, the patient had steadily deteriorated, losing weight and deteriorating mentally. Certification was imminent. He had been admitted to Sydney Hospital on August 3, 1956, with the diagnosis of acute intermittent porphyria. Two days after his admission he was inadvertently given an "A.P.C." gargle, which he swallowed; he was soon noticed to be acting queerly. Later that night he disappeared from the ward, and was finally persuaded to return to hospital from his home. His subsequent behaviour varied from pronounced drowsiness associated with incoherence and non-cooperativeness to pronounced activity associated with ideas of persecution. He considered that his bed was covered with germs and would madly smell his sheets and throw them away. His hypomania could be controlled by paraldehyde given intramuscularly. At times he was greatly preoccupied with his internal organs—for example: "has no liver and spleen"; "certain to have cancer of the stomach"; "bowels not open for 14 days"; "his bowels are gone". He was firmly convinced that he was being poisoned, and was very suspicious of the nursing staff and other patients, who "considered him a pariah". He was examined by Dr. A. T. Edwards, honorary psychiatrist, who considered that the patient appeared to be suffering from a depressive psychosis rather than paraphrenia. In spite of the porphyria, electroconvulsive therapy was the treatment most likely to be effective. Dr. Edwards suggested that the patient be sent to the Reception House for admission to a mental hospital, or that he be admitted to a private hospital. In spite of that advice, the patient's wife had insisted on taking the patient home on September 7, 1956.

Chronic Relapsing Pancreatitis Treated by Bilateral Splanchnicectomy.

DR. C. E. WINSTON, DR. P. H. GREENWELL and DR. J. V. L. COLMAN showed a married woman, aged forty years, who since 1941 had had a duodenal ulcer for which she had been treated medically in hospital on several occasions. Her previous operations included appendicectomy in 1930, bilateral salpingectomy in 1949, division of adhesions in 1952, and excision of a right submandibular gland for calculus in 1955. In May, 1955, she had been admitted to hospital with an exacerbation of severe epigastric pain radiating to the right and left hypochondria and through to the back; the pain awakened her at night and was not related to meals. Laparotomy was performed; no evidence of peptic ulceration was found in the stomach or duodenum on external examination. A hard mass was present, localized to the body of the pancreas, with areas resembling fat necrosis. A biopsy specimen was taken from the pancreas, and nothing further was done. Dr. A. A. Palmer, who made the pathological examination, reported the presence of active subacute inflammation of the pancreas with polymorphonuclear infiltration, disappearance of acini, dilatation of ducts and formation of young connective tissue. No necrosis was seen in the specimen examined.

In August, the patient was readmitted to hospital with an exacerbation of similar severe pain, which was greatly relieved by splanchnic block. In view of that finding, splanchnicectomy was advised.

In September, left thoraco-lumbar sympathectomy was performed. A remission of pain was obtained, but it later recurred, and tended to be localized more to the right side of the abdomen.

In January, 1956, right thoraco-lumbar sympathectomy was carried out; right hydropneumothorax was a complication. Since that operation the patient had not complained of the original pain.

Pseudo-Pancreatic Cyst.

Dr. Winston, Dr. Greenwell and Dr. Colman next showed a married woman, aged thirty-two years, who in January, 1956, had been referred from the Royal Hospital for Women, Paddington. During a routine examination of the patient after a miscarriage, a mass had been found in the left hypochondrium. Her only symptoms were thoraco-lumbar backache and aching in the left hypochondrium of two years' duration. A number of investigations were carried out. A blood count gave normal findings. The Kline test and the Casoni test both produced negative results. Excretion pyelography revealed a large, well-defined soft tissue mass in the left upper quadrant of the abdomen, displacing the kidney into the left sacro-iliac region, the renal shadow being distinct from the mass. A barium meal X-ray examination showed that the stomach and small bowel were displaced to the right and anteriorly; there was no intrinsic lesion. A barium enema was given, and an X-ray examination revealed depression and flattening of the splenic flexure, but no intrinsic lesion. Presacral pneumography and retrograde pyelography revealed a large extrarenal mass in the left upper quadrant of the abdomen, displacing the kidney downwards.

In March laparotomy was performed, a left upper paramedian incision being used. A large, thin-walled cyst occupying the lesser sac was found firmly adherent to the pancreas posteriorly and displacing the stomach down and to the right. Marsupialization to the parieties was performed. Healing was complete in eight weeks.

Squamous Carcinoma of Penis; Carcinoma of Head of Pancreas.

Dr. Winston, Dr. Greenwell and Dr. Colman finally showed a man, aged seventy years, who in June, 1952, had undergone partial amputation of the penis for squamous carcinoma. In December, 1952, a superficial group of right inguinal lymph nodes was excised and a right femoral hernia was repaired. Pathological examination of the lymph nodes revealed chronic inflammatory changes. In October, 1954, a block dissection of the left inguinal lymph nodes was carried out, with repair of a left femoral hernia. Pathological examination once more revealed chronic inflammatory changes. In December, 1954, the patient presented with painless progressive obstructive jaundice, thought to be due to metastatic deposits in the liver and the *porta hepatis*. Investigations did not assist in the making of a diagnosis. At laparotomy in January, 1955, a carcinoma of the head of the pancreas was found, with two large calculi in the gall-bladder. Pancreatico-duodenectomy was performed, and recovery was uneventful. Dr. A. A. Palmer, who made the pathological examination, reported that the tumour was an adenocarcinoma probably arising from a papilloma of the accessory pancreatic duct. No metastases were found in the lymph nodes.

In August, 1956, the patient was well and maintained his weight. His only symptom was occasional regurgitation of bile. He suffered from osteoarthritis of the right hip and spondylitis of the thoraco-lumbar part of the spine. The hæmoglobin value was 13·1 grammes *per centum*. An X-ray examination with a barium meal revealed a satisfactorily functioning gastro-enterostomy.

Ossifying Fibroma of Jaw.

DR. A. P. FINDLAY and DR. J. E. REIMER showed a small boy, whose mother had noticed a swelling of his left cheek four or five months prior to his admission to Sydney Hospital on January 12, 1956. The swelling was not painful, and there was no history of nose bleeding or discharge from the nose. The swelling had appeared to increase in size over the few days prior to the boy's admission to hospital. On examination of the patient, there was a swelling of the infraorbital region of the left cheek, which also involved the hard palate and the alveolar region of the left maxilla. On January 16 an examination of the skull revealed no lesion seen in the cranial vault or base. The tumour appeared to affect the left maxilla anteriorly. It was thought probably to be a fibroma or fibrosarcoma.

On January 23 the adenoids were removed and sent for pathological examination. A biopsy of the left maxilla was performed; the whole of the left maxillary antrum was occupied by what appeared to be a bony new growth. Pathological examination of tissue from the post-nasal space revealed large follicles with secondary centres. There was no evidence of lymphosarcoma, and the appearance suggested lymphoid hyperplasia. Examination of sections of bone from the anterior wall of the left maxillary antrum revealed well-formed bony spicules in a fibro-cellular matrix. This was thought to be an ossifying fibroma. There was no evidence of malignant change. On January 25 the patient was examined in the department of deep X-ray therapy (Dr. F. Duval). It was thought that deep X-ray therapy would be of little use in this case.

Excision of the left maxilla was performed on February 9 by Dr. A. P. Findlay. An incision was made along the lateral margin of the nose, across to the mid-line of the upper lip and along the mid-line of the hard palate. The external carotid artery was ligated. A flap was raised from over the left maxilla. The two processes of the zygoma were divided, the upper with an osteotome and the lower with a Gigli saw. The incision in the bone was carried along the inferior orbital margin and down the fronto-nasal process. The mucous membrane of the hard palate was cut with diathermy and the bone with an osteotome. The lateral pterygoid was divided with bone forceps. The maxilla

was then removed. The infraorbital plate came away with the maxilla. The inner surface of the flap was grafted with skin from the right thigh, and the flap was sutured back into position. The cavity was packed with "Vaseline" gauze.

The pathologist reported as follows on the specimen, which consisted of the entire left maxilla. Dense uniform cancellous bone filled the antrum except for a small cavity measuring 2 by 1·5 by 1 centimetre posteriorly. The abnormal bone was continuous with, and expanded, the alveolar process and the floor of the orbit. It was covered with a thin layer of compact bone. The plane of section through the palate appeared clear of abnormal bone, likewise the zygomatic process. Microscopic examination of the sections revealed ossifying fibroma similar to that in the biopsy specimen. On February 21 the patient was examined at the Dental Hospital by Professor A. J. Arnott. It was decided to construct an appliance to support the skin graft, and to prevent the falling in of the patient's cheek. On February 23 a prosthesis which built up the left infra-orbital region was fitted by the Dental Hospital. Several prostheses were made before a satisfactory one was obtained. The patient was discharged from hospital on April 10.

Gastrectomy for Anastomotic Ulcer.

Dr. Findlay and Dr. Reimer also showed a man, aged sixty-seven years, who had been admitted to Sydney Hospital on May 22, 1956, with a history of intermittent epigastric pain, relieved by powders and food, of two years' duration. During the eight weeks prior to his admission to hospital this pain had become much more severe. This latter pain commenced in the epigastrium and radiated to the right hypochondrium and right iliac fossa; it was not relieved by powders or food. "Amidone" tablets were necessary for its relief. The patient had also had perforated ulcer nine years earlier, followed by a gastro-enterostomy eight years earlier. He had been well for the next six years.

On examination, the patient was a thin, elderly man, in some pain. His tongue was moist and coated. His abdomen was thin, with three scars. Guarding was present in the epigastrium. No masses were palpable; extreme tenderness was present in the epigastrium. Rectal examination revealed greyish coloured stools.

A number of investigations were carried out. On May 22 the hæmoglobin value was 10 grammes per 100 millilitres. On May 24 an X-ray examination of the chest revealed chronic emphysema with scattered fibrous and calcified lesions in the apex of the left lung. On May 31 Graham's test was carried out; the gall-bladder contained small calculi, which were visible in all views. A barium meal X-ray examination on June 6 revealed a functioning anastomosis at a high level on the stomach, with some degree of contraction of the middle third of the stomach. There appeared to be an ulcer on the posterior wall at the lower end of the upper third. Portion at least of the barium left the stomach through the pyloric end. On June 16 a test for occult blood gave a negative result. On June 18 a gastroscopic examination was made; it revealed that a posterior gastro-enterostomy had been performed. There was a chronic peptic ulcer on the posterior wall just proximal to the stoma, with surrounding chronic gastritis.

A diagnosis of cholelithiasis and gastric ulcer was made, and a combined cholecystectomy and partial gastrectomy was performed by Dr. Findlay on June 21.

A mid-line incision six inches in length was made in the upper part of the abdomen. Dissection was difficult, owing to the presence of adhesions and fibrosis. The gall-bladder was removed, the cystic duct and artery being ligated with white silk. A large gastric ulcer was found near the previous anastomosis. The small intestine was divided on each side of the gastro-enterostomy, and the old anastomosis and the lower part of the stomach were then excised. The efferent loop of intestine was anastomosed to the stomach and the afferent loop to the jejunum. The duodenum was sutured in layers and the end of the stump was invaginated. A rubber drain was inserted and secured with a nylon stitch. The abdomen was closed with stainless steel wire. The skin was sutured with black silk.

The pathologist reported as follows on the organs removed. The first specimen was a portion of the stomach, with a piece of anastomosed jejunum attached. There was some degree of hour-glass constriction at the level of the anastomosis. The state of the neighbouring mucosa was difficult to determine, but fibrous thickening of the nearby stomach wall suggested a stomal ulcer. The second specimen was the gall-bladder, which contained five small stones. Examination of the mucosa revealed cholesterosis. Microscopic examination of sections of the stomach revealed chronic inflammation and fibrosis; in the gall-bladder chronic cholecystitis was present. During operation the patient was given whole blood. After the operation he was given packed red cells, fluids intravenously and hourly gastric aspirations.

The wound was dressed on July 2 and found to be clean and dry. Sutures were removed on July 2 and 3. He was discharged from hospital on July 28. At the time of his discharge the patient's pain was much less than on his admission. On his discharge the patient was receiving an ulcer diet, "Amphojel" as necessary and "Probanthine", one tablet three times a day.

Total Colectomy for Ulcerative Colitis.

Dr. Findlay and Dr. Reimer finally showed a man who had been admitted to Sydney Hospital on July 10 with a history of colicky abdominal pain and a mass in the right lower quadrant of the abdomen of one week's duration. The colicky pain was present every day just before his admission to hospital. The patient had suffered from ulcerative colitis for the past nineteen years. The last severe attack of abdominal pain had occurred in 1951, and during that attack blood and pus were passed *per rectum*. No blood or pus had been passed *per rectum* during this last attack.

On examination of the patient, a tender, smooth mass was felt in the right iliac fossa. It was approximately three inches in diameter and roughly spherical, and it seemed mobile. No tenderness or masses were felt on rectal examination. On July 24 an X-ray examination with a barium enema revealed advanced colitis, probably with ulceration. There was some narrowing of the ileo-caecal valve, apparently with some dilatation of the terminal portion of the ileum. A blood count showed that the hæmoglobin value was 9·0 grammes *per centum* and the leucocytes numbered 11,000 per cubic millimetre. The erythrocyte sedimentation rate was four millimetres in one hour. Examination of a stained film revealed anisocytosis, microcytosis and poikilocytosis; many target cells were present.

At operation, an oblique incision was made on the right side of the abdomen, the muscle layers were divided and the peritoneum was opened. Palpable masses were found in the caecum, the terminal portion of the ileum, the ascending colon and the transverse colon. The mesenteric glands were enlarged. A colectomy was performed, part of the pelvic colon being left. An ileostomy was performed through an incision in the left rectus muscle.

On July 27 the ileostomy appeared non-viable and gangrenous. A decision was made to resect part of the ileum. The right oblique incision was extended and nine inches of the terminal portion of the ileum were resected. The ileostomy was brought through the right oblique incision. A suprapubic drainage tube was inserted. Six inches of the terminal portion of the ileum were found to be gangrenous.

The pathologist reported as follows on the specimens removed at operation. The first specimen consisted of 15 centimetres of the transverse colon with a thickened wall, almost entirely infiltrated by a mucoid new growth of the *linitis plastica* type and showing mucus-filled cysts beneath the serous surface. The second specimen consisted of 25 centimetres of colon with a thickened wall. The mucosal surface was entirely covered by mamillations; a few were pedunculated and resembled polypi. At the centre there was an irregular linear shallow ulcer approximately two centimetres long on the mesenteric border. The third specimen consisted of 20 centimetres of ileum. There was an annular new growth two centimetres long protruding into the lumen and producing considerable obstruction. The remainder of the mucosa showed destruction of pattern and mamillation. The fourth specimen was a segment of jejunum showing no gross abnormality. Microscopic examination revealed the following findings. The lesion in the transverse colon was a mucoid adenocarcinoma; that in the ileum was a scirrhous adenocarcinoma. In the remainder of the bowel there were changes typical of chronic ulcerative colitis. A Davol bag was fitted to the ileostomy on August 6. The ileostomy had functioned well since operation except for two or three days (September 4, 5 and 6) when an obstruction developed. It was found that a stricture had developed at the ileostomy. The lumen was dilated with a finger, and that relieved the obstruction. The right oblique wound opened two weeks after operation—the skin sutures pulled out. The muscle layers did not open. At the time of the meeting the wound had healed almost completely by granulation and skin graft. The patient was still in hospital.

Surgery of the Parotid Gland.

DR. J. M. YEATES, in a demonstration of the surgery of the parotid gland, first showed a dissection of the facial nerve, with special reference to the arrangement of its branches in relation to the gland and to the facial veins. Dr. Yeates said that he believed all operations on the parotid gland should be prefaced by an exposure of the facial nerve, and that his preference was for finding the trunk first, using the posterior belly of the omo-hyoid muscle as a guide.

The first patient shown was a woman, aged thirty-five years, who had suffered for six years from recurrent attacks of severe pain and swelling in both parotid glands. Sialograms showed a condition of dilatation of the ducts. Excision of the left gland in June, 1955, gave such relief that the patient demanded similar treatment for the right side, even though she was warned that dryness of the mouth might be troublesome. The second operation was performed in February, 1956. After both operations the facial nerve took several months to recover fully. The patient denied even temporary dryness of the mouth.

The next patient was a woman, aged fifty-five years, who had complained of a swelling in the right parotid region for twelve years. At operation in December, 1956, the bulk of the gland was stripped from the branches of the facial nerve. Six months elapsed before perfect facial nerve function was restored. The pathological report stated that the gland contained an encapsulated tumour measuring four by three centimetres. Examination of sections showed this to be a pleomorphic adenoma (mixed tumour).

Maldescent of the Testis.

Dr. Yeates then showed three boys, all aged eleven to twelve years, whom he had treated during the previous twelve months for undescended testis. In each case the testis had lain high up in the inguinal canal, and could not be brought into the scrotum by traction. Two of the boys had been treated by applying the principle of fixation of the testis to the thigh (Thorek). In one case the result was poor, and in the other the testis resided about halfway down the scrotum. The third boy had been treated by the method described in Gross's text-book, with very pleasing results. The testis was identical in appearance with its fellow.

Dr. Yeates said that a really extensive dissection was the key to success. The *fascia transversalis* was incised, the inferior epigastric vessels were ligated, and the retroperitoneal space was exposed with Deaver's retractors. That exposure permitted mobilization of the internal spermatic artery almost to its origin from the aorta (some five inches above Poupart). The *vas deferens* could also be mobilized to its junction with the vesicle at the back of the bladder. After such dissection, the cord was so long that the testis could be dropped into the scrotum, and even the mild fixation described by Gross seemed superfluous. In the meticulous repair of the incised tissues, the inguinal canal was abolished, the cord running straight down to the scrotum, without deviation round the epigastric vessels. Dr. Yeates said that that operation was based on the sound surgical principle of relaxation of tissues, obtained by painstaking dissection, and contrasted sharply with the traction type of procedure. However, it was clear that future hernia would be likely if diligent repair of the abdominal wall was neglected.

Hæmatemesis Treated by Gastrectomy.

Dr. Yeates finally showed a woman, aged forty-seven years, who had been admitted to a medical ward on April 27, 1956, with the history that sudden severe hæmatemesis had occurred the previous day. The liver was palpable. After two days of medical treatment the bleeding continued unabated. At operation on April 29 the liver, stomach and duodenum appeared normal. When the lesser sac was opened, and thus a bimanual grasp of the lesser curve of the stomach was obtained, a slight induration appeared to be present. On that somewhat doubtful evidence, gastrectomy was performed. Examination of the specimen revealed a small ulcer with a partly thrombosed central artery.

Anaplastic Carcinoma of the Ovary with Ascites.

DR. H. K. PORTER and DR. A. A. MOON showed a married woman, aged sixty-two years, who had had four children. She had been admitted to Sydney Hospital in August, 1955, with a history of vaginal bleeding of three days' duration and of intermittent cramp-like pains in the lower part of the abdomen for one year. The menopause had occurred in 1944. In 1938 she had undergone right radical mastectomy for scirrhous carcinoma. On examination of the patient, an abdominal mass was present extending from the pelvis to just below the level of the umbilicus. There were signs of free fluid in the abdomen. Vaginal examination suggested that the mass was continuous with the uterus and was not fixed.

At operation on September 7, straw-coloured fluid was aspirated from the peritoneal cavity. The abdomino-pelvic mass measured six by six by eight inches, and was cystic and multiloculated. It was adherent to the uterus, bladder, omentum and small bowel. The tumour had its origin in the right ovary. Secondary tumour nodules were present in the omentum and on the bladder wall. The tumour mass was removed. Deep X-ray therapy was commenced on September 25.

In February, 1956, the abdomen became swollen, and the patient was readmitted to hospital for paracentesis. Since then she had returned to hospital at intervals of approximately three weeks for aspiration of free fluid in the abdomen. Pathological examination showed the tumour to be an anaplastic carcinoma.

Papillary Adenocarcinoma of the Ovary with Ascites.

Dr. Porter and Dr. Moon then showed a married woman, aged fifty-six years, who had had two children. She had undergone total hysterectomy in February, 1949, for a large fibromyoma. In January, 1956, she suffered from headache, nausea, abdominal discomfort and mild diarrhœa, and a provisional diagnosis of infective hepatitis was made in the country. The symptoms settled in March, but soon afterwards her abdomen became progressively enlarged. *Paracentesis abdominis* was performed in the country, and a hard mass could then be felt in the lower part of the abdomen and the pelvis. She was then referred to Sydney Hospital with the provisional diagnosis of carcinoma of the ovary.

The patient was admitted to hospital on July 23 with gross abdominal distension and signs of ascites. There was a hard mass filling most of the pelvis, but not attached to the pelvic floor; it was slightly mobile. The mass extended into the abdomen on each side of the mid-line.

At operation on July 25, 30 pints of blood-stained fluid were aspirated from the peritoneal cavity. The left ovary was replaced by a friable papillary tumour, which had a pedicle and could be removed completely. A neoplasm of the right ovary almost filled the pelvis, but could be delivered and removed intact. There were no visible or palpable masses in the omentum, bowel, liver or regional lymph nodes. Pathological examination showed the tumour to be a papillary adenocarcinoma. Convalescence was uneventful. Deep X-ray therapy was commenced on August 6 and finished on August 29. The patient was discharged from hospital on September 4.

Besnier's Prurigo Treated with "Decortisyl".

DR. EWAN MURRAY-WILL showed a male patient, aged nineteen years, who had had widespread eczema all his life, associated with asthma and hay fever. The disease had frequently been generalized, and at one period was so severe that he was kept in hospital for three years. There had been periods of slight improvement, but the patient had never been free from eczema, and usually the itching had been intense and had caused great interference with sleep. The father and sister suffered from asthma, the mother from hay fever.

When he was first examined on his admission to Sydney Hospital on August 18, 1956, the patient had generalized eczema, with considerable lichenification of the flexures of the elbows, wrists and knees. There was scaly erythema of the face, the neck and the greater part of the trunk and extremities, with numerous excoriations.

"Decortisyl" therapy was instituted, oral doses of 60 milligrammes per day being given for the first three days, then 50 milligrammes per day for three days. Dr. Murray-Will said that the dose had been subsequently reduced by five milligrammes per day, and the patient was now having 20 milligrammes per day. Itching had ceased completely within the first few days of treatment, and the eczema had diminished rapidly in extent and in lichenification. Recently a mild acneiform eruption had developed on the upper part of the trunk as the result of the "Decortisyl" therapy, but it was not causing the patient any inconvenience. *Pasta Picis* (3%) had been prescribed for local application, but that produced tar folliculitis and was discontinued. Zinc oil was substituted for a few days, and subsequently 1% hydrocortisone cream was applied to the flexures of the elbows and knees. Superficial X-ray therapy (100*r* unfiltered) had been given to the flexures of the elbows and knees on August

24 and 31. The patient said that his skin had never been so clear as it was at the time of the meeting, and he felt well and had no itch at all.

Glomus Jugulare Tumour.

DR. ARTHUR REVAI showed a female patient, aged forty-five years, who had been suffering from earache, buzzing noise in the ear and deafness of the left ear for the last ten years on and off. On three occasions removal of polypi from the external meatus was accompanied by considerable hæmorrhage, and followed by rapid reformation of the tumour. Radical tympano-mastoidectomy was performed on March 7, 1956, with eradication of the tumour masses which seemed to be arising from the floor of the tympanic cavity (roof of the jugular bulb) and the region of the tympano-pharyngeal tube ostium. The expected profuse loss of blood was counterbalanced by blood transfusion during and after operation. Four weeks after the operation deep X-ray treatment was given, to a total dosage of 3000*r* over two weeks. At the time of the meeting, six months after the operation, there was no trace of any recurrence of the neoplasm.

Dr. Revai said that normally two or three jugular bodies were found in each temporal bone. They were small structures, 0·2 by 0·5 millimetre in size, and were situated along the tympanic branch of the ninth nerve (Jacobson's nerve)—from the jugular fossa to the promontory—and along the auricular branch of the tenth nerve (Arnold's nerve)—from the jugular fossa to the external meatus. Not infrequently a twig from the last-mentioned nerve travelled along the facial canal. Location of the bodies was of importance to explain the variety and severity of the symptoms. Pathological examination of the tumour showed it to consist of masses of blood vessels of capillary and pre-capillary size, and in between those vessels there were numerous epitheloid cells. Metastasis never occurred, but from a clinical point of view the tumour was malign owing to the pressure it exerted on vital structures. Jugular body tumours were not rare; at the present time they were encountered and recognized more often than previously. Every aural polypus whose removal caused considerable bleeding should arouse suspicion. Radical operation followed by deep X-ray treatment was the required procdure to deal with those tumours.

Conversion Hysteria.

DR. OSCAR R. SCHMALZBACH showed a married woman, aged thirty-eight years, who had been referred to him by the ear, nose and throat clinic because of her inability to speak in a normal voice for one week. The loss of voice had come suddenly, and the patient had since spoken in a whisper. Ear, nose and throat examination revealed no cause for the complaint. The patient gave during the psychiatric interview the following history, which was substantiated by a few details taken by the social worker, Miss Tracey, in a separate interview. The patient said that she came from a happy family, being one of nine children. Her schooling was average, and although she liked school she left at the age of about fourteen years, and started to work in the Postmaster-General's Department. She intended to become a nurse, but never fulfilled her ambition. She left post-office work after a few months, and remained in her next employment for about three years, working as a labourer in a factory packing and labelling parcels. She married when aged eighteen years, after knowing her husband since her early adolescence. She admitted that her married life was not necessarily a happy one because of the gambling interests of her husband, which did not bring a feeling of financial security. The patient had to live with her parents for a while during the financial depression when her husband was unemployed. She had three daughters and one son, their ages ranging from nineteen to thirteen years respectively. There had been housing and financial difficulties, the patient's husband had had several illnesses, and the patient had had difficult confinements sometimes complicated by secondary anæmia. The accommodation which the family had had for thirteen years was too small for their needs, and the eldest daughter had to board with neighbours. During the last eight months the patient's husband had not worked because of dermatitis, and was receiving a social services allowance. The patient was financially helped by her children and tried to carry on her domestic duties in spite of the difficulties. On being further questioned, the patient described her husband's attitude towards her. She said that he was often bad-tempered, and was suspicious, accusing her of unfaithfulness, although it had never occurred, and she believed that it was he who in the early years of their married life was unfaithful. He was unsociable and disliked friends, and on rare occasions when they had guests at their home he would give expression to his dissatisfaction. Their sex life was not satisfactory to the patient. She resented it, and sometimes felt "like screaming" when some sex demands were made by her husband. That feeling of abnegation in their relationship the patient had experienced for the last few years. One year prior to the meeting she had become depressed after the death of her father, to whom she felt very much attached.

On examination of the patient, no neurological abnormality was found. The patient was pale, but she said that that was her complexion. She spoke subvocally, and whispered her story. She was depressed, worried and upset. She denied having delusions and hallucinations of any kind. She seemed tidy in appearance and cooperative, and her orientation for time and place was good.

In summing up her case, Dr. Schmalzbach said that he considered that the patient suffered from a depressive state, in which her sudden loss of voice was a conversion hysterical expression of powerlessness against her life situation, particularly that related to her sex life with her husband. Psychosomatically that sudden loss of voice for which there was no organic basis was called in the German language *"Organ-sprache"*—the speech of an organ. In other words, in the sudden development of a functional aphonia the patient gave expression to her revolt against the sexual advances of her husband and against her life situation generally. She said that in such a situation she would like to scream; but instead of screaming she did the opposite, developing aphonia in accordance with the old Latin saying *"cum tacent clamant"*, which meant "they scream whilst being mute". That conversion was the basis of the patient's symptom.[1]

Huntington's Chorea.

DR. I. A. LISTWAN showed a female patient, aged forty-one years, who had been married twice and had two children. She had been well until four months previously, when she noticed occasional shaking of her head and spasms of the face and hands. She had no other complaints. Her attention had been drawn to her symptoms because they had appeared before in her family. Her father had become ill with similar symptoms at the age of forty-seven years, and he died ten years later with the diagnosis of Huntington's chorea. A brother, aged fifty-two years, had been in a mental hospital for several years with the same diagnosis. Another middle-aged brother, who was out of touch with the family, was thought to have the same disease. The patient's mother had been in a mental hospital for many years. The patient's daughter had had blackouts since the age of one year; her son enjoyed normal health. On closer inquiry, the patient said that she had recently noticed a deterioration in her concentration and memorizing power. She had difficulty in walking up steps and in holding a glass of water. She had also noticed a deterioration in her speech and difficulty in finding words. She had recently become depressed, and because of that had lost interest in social relations. There were no factors of importance in her personal history. She had completed her primary education and seemed to enjoy a normal school life. She had excelled at sport, and was more interested in that than in lessons. Until her marriage she was successfully employed as a buyer in a millinery business.

On examination of the patient, occasional nodding of her head and an inappropriate smile were noticed. She had impaired coordination in her hands, with some difficulty in buttoning a coat, fixing the shoe-laces and holding a glass of water. Her writing was slightly out of line and the letters were of different size. She wrote better with a ball-point pen than with an ordinary pen. She walked with a limp and slight ataxia. No other neurological signs were detectable. No abnormality was found in the X-ray film of the skull, and the blood count was normal. An electroencephalogram showed no organized *a* rhythm, and the dominant rhythm was a fast 18 to 30 cycles per second rhythm in the central, temporal and occipital areas. That was the type of record seen in states of nervous tension. There was no indication of focal abnormality. The comment was made that the electroencephalogram was normal in about half the cases of Huntington's chorea. The patient's intelligence quotient on the Bellevue-Weschler scale was estimated as: general 72 (borderline); verbal 84 (dull normal); practical 70 (mental defective); the deterioration index was 79%.

Dr. Listwan said that the patient was shown because of the rarity of the condition and because of the typical syn-

[1] One week after the meeting the patient reported that her voice had suddenly returned. It had gone away again after an argument with her husband, but had come back again, she hoped permanently.

drome. Huntington's chorea usually started in middle age, developed for years, was more common in men than women, was hereditary, and carried a tendency to mental deterioration. The inheritance was dominant and never missed a generation. Neurologically the disease was characterized by fidgettiness, grimacing, twisting, nodding or wriggling of the head, jerky gait, unmotivated smile and explosive speech. Mentally it was characterized in the first stage by emotional changes (irritability, tempers), followed later by character changes (eccentricity, alcoholism, sexual abnormality) and depressive moods (self-accusing, impulsive and suicidal). Unfortunately, it was followed later by an organic type of dementia with deterioration of memory (power of recall), disorientation, and poverty of ideas. The pathology was characterized by frontal cortical brain atrophy, frontal-rolandic leptomeningitis, internal hydrocephalus, gliosis and vascular changes. The prognosis was poor. It ended inevitably in ten to fifteen years in complete dementia and its sequels. There was no treatment for the condition, and marriage of affected persons should be banned.

Breast Carcinoma Controlled by Surgery, X-Ray Therapy and Hormones.

DR. F. DUVAL, DR. P. M. C. CORLETTE, DR. R. C. SCOBIE and DR. SYLVIA BRAY showed three patients to illustrate the control of carcinoma of the breast by means of surgery, X-ray therapy and hormones.

The first patient was aged forty-four years. In December, 1951, she had undergone a radical mastectomy for an advanced stage I carcinoma of the breast. In December, 1955, she reported with metastases in the neck of the right femur; deep X-ray therapy was given to the area, and ablation of ovarian function by deep X-ray therapy was carried out. In May, 1956, she was found to have metastases in the skull, and testosterone therapy was begun, with relief of her pain. Her general condition was good.

The second patient was aged thirty-nine years. In October, 1953, she had undergone left radical mastectomy for advanced stage II carcinoma of the breast. A post-operative course of X-ray therapy was given to the gland drainage areas. In November, 1954, she was found to have bony metastases in the lumbo-sacral part of the spine and the bony pelvis, and testosterone therapy was given. In March, 1955, further bony metastases were found in the ribs and in the lumbar part of the spine. In December, 1955, ovarian function was ablated by deep X-ray therapy. Since her condition was not controlled by that treatment, bilateral oophorectomy was performed in November, 1955, and on microscopic examination carcinomatous deposits were found in both ovaries. The patient had been receiving male hormones since March, 1955, and was in fairly good general health. Her condition had been fairly well controlled by that treatment. She did not wish to undergo adrenalectomy at the present time, so she was continuing to take non-virilizing male hormone tablets.

The last patient shown had been aged forty-two years in 1948, when she underwent left radical mastectomy for medullary carcinoma; the stage of the growth could not be ascertained. In November and December, 1948, she was given a course of post-operative irradiation to the gland drainage areas. In July, 1950, skin nodules appeared, and that process continued until early in 1954. They were treated by superficial X-ray therapy, and responded when they were small and localized; but in February, 1954, they began to spread quickly. Oral treatment with testosterone was begun at that time. In January, 1955, involvement of the right breast and axillary glands was found. In July, 1955, an extensive *"en cuirasse"* condition appeared; it was controlled by an increase in the dosage of testosterone by the intramuscular injection of 100 milligrammes per week, 150 milligrammes per day being taken by mouth. In April, 1956, the patient consented to undergo bilateral adrenalectomy. On microscopic examination, one ovary was found to contain metastatic carcinoma. Within four weeks of the operation, the whole of the *"en cuirasse"* condition had disappeared. The right breast had softened and become mobile on the chest wall, the hirsuties had decreased, and the patient's general condition had improved. Since April she had been living a normal life, and there was no radiographic or clinical evidence of carcinoma.

Orthopædic Conditions.

DR. B. BLOCH, on behalf of the orthopædic team (DR. W. S. L. STENING, DR. A. I. RHYDDERCH and himself) showed several patients with orthopædic conditions.

Intramedullary Insertion of Kuntschner Nail in Fractured Shaft of Femur.

Three patients were shown to illustrate the use of the Kuntschner nail in the treatment of fractures of the femur. The first was a man, who had had an ununited transverse fracture of the shaft for two years. He had been immobilized in a traction apparatus and later a plate had been applied. He was admitted to Sydney Hospital, where a Kuntschner nail was inserted; union occurred. The second patient was a man with a long oblique fracture, who had been sent down from the country. A very large hæmatoma was present. A nail was inserted. The third patient was a man who had sustained a transverse fracture of the shaft of the femur. Dr. Bloch commented that the three cases illustrated the advantages of the Kuntschner nail, and the use of early ambulation with crutches and calipers, knee-joint movement being satisfactory.

Segmental Fractures of the Tibia Treated with Eggers's Plate.

Dr. Bloch next showed a married woman, who had been admitted to hospital from the casualty department on October 8, 1954, with segmental fractures of the left tibia and fibula and a fracture of the pelvis. On December 10 the fractures of the tibia were plated with double Eggers's plates. The caliper had been discarded in December, 1955. Union at the time of the meeting was very firm.

Carpal Tunnel Syndrome.

The next patient shown was a married woman, who had suffered from pain in the neck and in the left forearm and hand. The pain in the left hand was very severe and kept her awake at night. Operation was undertaken in April, 1956. The median nerve was explored, and the retinaculum overlying the carpal tunnel was divided. At the time of the meeting the patient was free of symptoms.

Dislocation of the Hip with Sciatic Nerve Injury.

The final patient shown was a man, who had fallen off a tram on September 8, 1955. He had sustained a compound dislocation of the right hip, and the head of the femur was presenting through an open wound in the right buttock. A complete sciatic nerve lesion was present. The dislocation of the hip was reduced, and the patient was put in a plaster spica in the position of wide abduction. During the next ten weeks the amount of abduction was decreased in successive plaster spicas. The spica was removed after seventeen weeks, and he was given a long leg weight-bearing caliper. Dr. Bloch commented that there had been remarkable recovery of the sciatic nerve, and that no avascular necrosis of the femoral head had occurred.

Almoner's Department.

The almoner's department presented a project on "Medical Social Work in Action". A series of eight illustrated posters, together with an explanatory leaflet, were prepared, giving details of social disabilities affecting patients referred to the department for assistance during 1955 and an outline of the services offered to them.

Out of the Past.

In this column will be published from time to time extracts, taken from medical journals, newspapers, official and historical records, diaries and so on, dealing with events connected with the early medical history of Australia.

THE PRESIDENT AND GENTLEMEN OF THE COURT.[1]

[From the Wentworth Papers.]

I HAVE pleaded not Guilty to the charge that has been brought against me. A charge which accuses me His Majestys Senior Assistant Surgeon at Parramatta of Contempt and Disobedience of the Orders of my Superior Officer in refusing to receive two Government servants into the Hospital there.

Gentlemen I know this is not the proper time for me to enter on my defence to this charge but I am induced by

[1] From the original in the Mitchell Library, Sydney.

motives which are of the utmost Importance to my future welfare, the consequences of which are to me incalculable, thus publickly to call on the Gentleman who appears as my Prosecutor on this Occasion, and tell him on this early stage of the Prosecution that if he is acquainted with a single Instance wherein I have taken any person into the Hospital to serve my own private purposes, or refused to receive any sick person from motives of personal resentment or a wish to impede the Kings Service, or if during the seventeen years I have been in this Colony, he knows of one case wherein I have neglected the Duties of my Situation or treated with Inhumanity the sick entrusted to my care or that I have excused any out patient either man or woman from public Labour, even for a single day with a view to my own Advantage or private Gratification, or if he can prove that I have converted to my own use or other improper purposes any part of the Necessaries committed to my charge for the use of the Sick, he will this day be guilty of a neglect of that Duty he owes to the King, to his Country and what I consider of equal Importance—His Duty to Himself, if he does not now bring it forward.

D. WENTWORTH, Assist. Surgeon.

Sydney,
New South Wales,
July 18th, 1807.

[D'Arcy Wentworth was charged by Governor Bligh with employing servants of the Crown in labour on his own grounds and in looking after his own stock, and with employing for his private emolument settlers' servants who had been sent into hospital sick. After Bligh's removal from office, Wentworth was on February 17, 1808, tried by General Court Martial and found not guilty on both charges.]

Naval, Military and Air Force.

APPOINTMENTS.

THE following appointments, changes *et cetera* have been promulgated in the *Commonwealth of Australia Gazette*, Number 76, of December 20, 1956.

NAVAL FORCES OF THE COMMONWEALTH.

Citizen Naval Forces of the Commonwealth.

Royal Australian Naval Reserve.

Promotion.—Surgeon Lieutenant Desmond Albert Cooper is promoted to the rank of Surgeon Lieutenant-Commander, dated 9th October, 1956.

ROYAL AUSTRALIAN AIR FORCE.

Permanent Air Force.

Medical Branch.

The following Flight Lieutenants are promoted to the rank of Squadron Leader: A. V. Coleman (033029) (Temporary Squadron Leader), J. R. Harrison (013663) (Acting Squadron Leader).

Australian Medical Board Proceedings.

NEW SOUTH WALES.

THE following additions and amendments have been made to the Register of Medical Practitioners for New South Wales in accordance with the *Medical Practitioners Act*, 1938-1955:

Registered medical practitioners who have complied with the requirements of Section 17 (3) and are registered under Section 17 (*a*) of the Act: Court, John Maurice, M.B., B.S., 1953 (Univ. Melbourne); Handley Donald Alfred, M.B., B.S., 1952 (Univ. Adelaide); Steel, James Henry, M.B., B.S., 1950 (Univ. Queensland).

Registered medical practitioners who have complied with the requirements of Section 17 (3) and are registered under Section 17 (*b*) of the Act: Binks, John Burcham, M.B., Ch.B., 1947 (Univ. Birmingham); Burry, John Neilson, M.B., Ch.B., 1950 (Univ. Aberdeen); Clarke, James Alan, M.B., Ch.B., 1943 (Univ. Glasgow); Dawes, Joan Margaret Stevenson, M.B., Ch.B., 1948 (Univ. Glasgow); Dawes, Peter, M.R.C.S., L.R.C.P., 1940 (Univ. London); de Zilva, Edith Maureen,

DISEASES NOTIFIED IN EACH STATE AND TERRITORY OF AUSTRALIA FOR THE WEEK ENDED DECEMBER 15, 1956.[1]

Disease.	New South Wales.	Victoria.	Queensland.	South Australia.	Western Australia.	Tasmania.	Northern Territory.	Australian Capital Territory.	Australia.
Acute Rheumatism	1	2(1)	3	1(1)	1	..	..	..	8
Amœbiasis	..	..	..	..	..	..	..	..	..
Ancylostomiasis	..	..	..	..	..	..	..	..	..
Anthrax	..	..	..	..	..	..	..	..	..
Bilharziasis	..	..	..	..	..	..	..	..	..
Brucellosis	..	..	..	..	..	..	..	..	..
Cholera	..	..	..	..	..	..	..	..	..
Chorea (St. Vitus)	..	..	..	..	..	..	..	..	..
Dengue	..	..	..	..	..	..	..	..	..
Diarrhœa (Infantile)	2	3(2)	3(3)	..	..	..	..	..	8
Diphtheria	1(1)	1(1)	..	..	2(1)	..	..	..	4
Dysentery (Bacillary)	..	..	2(2)	1	..	..	3	..	6
Encephalitis	..	3(3)	..	..	..	..	..	..	3
Filariasis	..	..	..	..	..	..	..	..	..
Homologous Serum Jaundice	..	..	..	..	..	..	..	..	..
Hydatid	..	..	..	..	..	3	..	..	3
Infective Hepatitis	63(20)	29(15)	..	12	1	7	1	..	113
Lead Poisoning	..	..	1	..	..	..	..	..	1
Leprosy	..	..	..	..	1	..	1	..	2
Leptospirosis	..	..	1	..	..	..	..	..	1
Malaria	..	..	2(1)	..	..	..	..	..	2
Meningococcal Infection	1(1)	4(2)	..	..	..	2(1)	..	..	7
Ophthalmia	..	..	..	..	..	..	..	..	..
Ornithosis	2(2)	..	..	..	..	..	..	..	2
Paratyphoid	..	..	..	..	..	..	..	..	..
Plague	..	..	..	..	..	..	..	..	..
Poliomyelitis	6	1	1(1)	2(1)	..	..	..	..	10
Puerperal Fever	..	..	1	..	..	..	..	..	1
Rubella	..	42(28)	1(1)	82(56)	..	..	..	..	125
Salmonella Infection	..	..	..	..	1(1)	..	..	..	1
Scarlet Fever	7(5)	15(8)	6(3)	6(2)	1(1)	2	..	..	37
Smallpox	..	..	..	..	..	..	..	..	..
Tetanus	..	1	..	..	..	..	..	..	1
Trachoma	..	..	..	..	..	..	..	..	..
Trichinosis	..	..	..	..	..	..	..	..	..
Tuberculosis	38(27)	20(13)	14(4)	8(6)	11(8)	7(5)	1	..	99
Typhoid Fever	..	..	..	..	..	..	..	..	..
Typhus (Flea-, Mite- and Tick-borne)	..	..	..	..	2(2)	..	..	..	2
Typhus (Louse-borne)	..	..	..	..	..	..	..	..	..
Yellow Fever	..	..	..	..	..	..	..	..	..

[1] Figures in parentheses are those for the metropolitan area.

M.B., B.S., 1948 (Univ. Ceylon); Fisher, Anne Sheila, M.B., B.S., 1954 (Univ. London); Hudspith, John Hendley, M.B., B.S., 1954 (Univ. Durham); Kramer, Harold, M.B., Ch.B., 1940 (Univ. Capetown); MacLachlan, Hamish Alistair, M.B., Ch.B., 1941 (Univ. Glasgow).

QUEENSLAND.

THE following have been registered, pursuant to the provisions of Section 19 (1) (*a*) and (*d*) of *The Medical Acts*, 1939 to 1955, of Queensland: Turner, Harold Montagu, L.R.C.P., M.R.C.S. (London), 1953; Nowell, Thomas Algernon, M.B., Ch.B., 1944 (Univ. Manchester).

The following additional qualifications have been registered: Trenerry, Frederick, M.C.P.A., 1956; Sullivan, John Joseph, M.C.P.A., 1956; Galea, Eugene George, M.R.C.P. (London), 1955; McKelvie, Ian James, M.R.A.C.P., 1956; Graff, John Frederick Hubert, F.R.C.S. (England), 1955; Hobson, David Lennox, M.R.A.C.P., 1953, M.R.C.P. (London), 1956.

The undermentioned persons (all having the qualifications M.B., B.S., 1956 (Univ. Queensland)) have been granted limited registration as medical practitioners, pursuant to Section 20 (3) of *The Medical Acts*, 1939 to 1955: Andonov, Nikola Dimitrijevic; Backstrom, David Leon; Broadbent, Geoffrey Donald; Buchanan, Thomas Blair; Carey, Justin Daniel; Carroll, Kenneth John; Casey, John Howard; Cave, Frank Maurice; Cheong, Mervyn; Chong Wah, George; Claxton, Colin James; Cleary, Judith Ann; Dewar, Malcolm Thomas; Douglas, William Alexander Charles; Farmer, Barry Edgar; Fielding, Avis Marie; Finlay, Frances Ann; Forrest, David James; Gallagher, Andrew Godfrey Patrick; Graff, Robin Roy Roger; Green, Clodagh; Hartmann-Johnsen, Olaf Johan; Herron, John Joseph; Hill, John Brian; Hodges, Roy John; Holmes, Noel Clarkson; Hurley, William Patrick James; Hurst, Ronald Gordon; Johnston, Stuart Ruddle; Lithgow, David Roy; Luck, Victor Roy; Moo, Edgar; McCaffrey, John Francis; McIntyre, Kenneth John; Nicoll, David Alexander; O'Dwyer, Patrick Francis; O'Neill, John Michael; Packer, Ronald Andrew; Parker, William Robert; Payne, Dorothy Barbara; Pegg, Stuart Phillip; Rainnie, Ronald George; Ramm, Ronald Arthur; Sheil, Ainslie Glenister Ross; Simpson, David Graham; Thomas, Thomas Powell; Thomson, Ronald Leslie; Thrift, Errol George; Todd, Bryan Edmund; Tucker, Arthur Douglas; Unwin, John Robertson; Waller, John Powell; Wilson, Charles Roy; Whitchurch, Charles Walter; Withers, Hubert Rodney; Gregg, Margaret Mary.

Honours.

NEW YEAR HONOURS.

THE following medical practitioners have been included by Her Majesty the Queen in the New Year Honours list:

Dr. John George Hunter has been created a Companion of the Most Distinguished Order of Saint Michael and Saint George.

Dr. Percival Landon Bazeley, O.B.E., and Dr. Ralph Whishaw have been created Commanders of the Most Excellent Order of the British Empire (Civil Division).

Dr. Henry John Chadwick Hanrahan and Dr. George Simpson have been created Officers of the Most Excellent Order of the British Empire (Civil Division).

Surgeon Rear-Admiral Lionel Lockwood, M.V.O., D.S.C., R.A.N., has been created a Commander of the Most Excellent Order of the British Empire (Military Division).

Medical Appointments.

Dr. Christina R. Trood has been appointed a medical officer in the Mental Hygiene Branch, Department of Health, Victoria, pursuant to the provisions of Section 18 (2) of the *Mental Hygiene Authority Act*, 1950.

Dr. Peter H. Sherwood has been appointed a medical officer in the Mental Hygiene Branch, Department of Health, Victoria, pursuant to the provisions of Section 18 (2) of the *Mental Hygiene Authority Act*, 1950.

Dr. R. S. Neilsen has been appointed government medical officer at Inglewood, Queensland.

Dr. Josiah Mark Bonnin has been appointed an honorary physician at the Royal Adelaide Hospital.

Dr. Richard Edmund Britten Jones has been appointed an honorary assistant physician at the Royal Adelaide Hospital.

Dr. David Charles Thompson has been appointed an honorary clinical assistant to the medical section at the Royal Adelaide Hospital.

Deaths.

THE following deaths have been announced:

STEELE.—Kenneth Nugent Steele, on December 19, 1956, at Adelaide.

SMEATON.—Bronte Smeaton, on December 20, 1956, at Adelaide.

LITTLEWOOD.—Frank Ernest Littlewood, on December 27, 1956, at Melbourne.

ROSS.—Colin Chisholm Ross, on December 31, 1956, at Sydney.

NEWMAN-MORRIS.—John Newman-Morris, on January 3, 1957, at Melbourne.

Diary for the Month.

JAN. 14.—Victorian Branch, B.M.A.: Finance, House and Library Subcommittee.
JAN. 15.—New South Wales Branch, B.M.A.: Medical Politics Committee.
JAN. 17.—Victorian Branch, B.M.A.: Executive Meeting.
JAN. 17.—South Australian Branch, B.M.A.: Council Meeting.
JAN. 23.—Victorian Branch, B.M.A.: Council Meeting.
JAN. 25.—Queensland Branch, B.M.A.: Council Meeting.

Medical Appointments: Important Notice.

MEDICAL PRACTITIONERS are requested not to apply for any appointment mentioned below without having first communicated with the Honorary Secretary of the Branch concerned, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

New South Wales Branch (Medical Secretary, 135 Macquarie Street, Sydney): All contract practice appointments in New South Wales.

Queensland Branch (Honorary Secretary, B.M.A. House, 225 Wickham Terrace, Brisbane, B17): Bundaberg Medical Institute. Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY HOSPITAL or position outside Australia are advised, in their own interests, to submit a copy of their Agreement to the Council before signing.

South Australian Branch (Honorary Secretary, 80 Brougham Place, North Adelaide): All contract practice appointments in South Australia.

Editorial Notices.

MANUSCRIPTS forwarded to the office of this journal cannot under any circumstances be returned. Original articles forwarded for publication are understood to be offered to THE MEDICAL JOURNAL OF AUSTRALIA alone, unless the contrary be stated.

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